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THE MEASUREMENT OF MORTALITY IN AUSTRALIA.

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The death rate is a fact, anything beyond this is an inference.—WILLIAM FARR.

IN a series of articles, I am attempting to analyse the mortality experience of the population of Australia, as recorded in the official publications of the Commonwealth, making special reference to the more recent years since Federation, for which a dissection by cause for Australia as a whole is available. It is important, when considering the mortality from individual causes, to have for comparison a more general study of the mortality from all causes. Further, we may hope to avoid repetition elsewhere by considering in detail here certain aspects of the computation and interpretation of mortality rates.

THE PERIODS CHOSEN FOR STUDY.

The deaths for Australia as a whole by age and sex and cause are available in convenient form only for 1908 and subsequent years. At the commencement of the study the most recent year available was 1945, and so the experience that is to be analysed extends over thirty-eight years and comprises some two hundred and twenty-six million years of life, one hundred and fifteen million years being experienced by males and one hundred and eleven million by females. The experience is, therefore, comparable in magnitude with that of Dublin and Lotka (1937), which consisted of three hundred and forty-six million years of life lived by persons insured with the Metropolitan Life Assurance Company, between the ages of one year and seventy-five years. In our study we shall make comparisons, not between the experiences of individual calendar years, but between those of groups of years or periods. In the choice of the periods we have as far as possible kept to the decennia customary in demographic work. There are thus three complete decennia and two shorter periods for study. In combining the experience of the years in this way we shall be losing much information of interest in the case of the epidemic diseases; but we cannot hope, in a survey of this kind, to do more than bring out the broad tendencies or trends in the death rates from such causes. On the other hand, in the diseases of a non-epidemic nature much is gained by considering these longer periods, for we are able thereby to reduce the sampling errors of the rates as measured by the coefficient of variation, and to attempt finer dissections of the disease itself—for example, we may consider the deaths of cancer by site. For, if we attempted this analysis by site for individual calendar years, we should find that the sampling errors would dominate the discussion. In fact, as will be seen at a later date, we shall find it necessary when analysing the deaths from the relatively common cancers peculiar to the female to use only two periods for comparison—namely, 1908 to 1930 and 1931 to 1945.

DEFINITIONS OF A DEATH.

In general there is little ambiguity about the fact of death or its registration; but there are three instances in which some care must be taken when international comparisons are being made. Firstly, certain deaths by violence may be presumed to have occurred but cannot be registered for legal reasons. This group is usually unimportant. Secondly, deaths in war time may not be

TABLE I.

The Measurement of Mortality in Australia.

Deaths (All Causes) in Australia by Age and Sex for Certain Periods.

Period.	Sex.	Deaths in the Age Groups (Years).										
		0 to 4.	5 to 14.	15 to 24.	25 to 34.	35 to 44.	45 to 54.	55 to 64.	65 to 74.	75 and Upwards.	Unclassified.	Total.
1908 to 1910 ..	M.	18,670	2512	4144	4730	6424	8901	8427	11,357	12,928	207	78,300
1911 to 1920 ..	M.	67,389	9885	14,374	20,737	24,548	34,712	41,019	42,322	49,560	575	305,121
1921 to 1930 ..	M.	55,761	9486	13,182	16,687	23,886	33,689	51,340	59,495	54,077	409	318,012
1931 to 1940 ..	M.	35,425	8499	12,934	14,568	21,354	38,406	57,774	81,835	82,584	177	353,556
1941 to 1945 ..	M.	18,557	3470	4349	5073	9037	19,873	35,818	46,247	55,346	35	197,805
1908 to 1910 ..	F.	14,934	2171	3853	4622	4894	4974	5020	7931	9454	35	57,888
1911 to 1920 ..	F.	53,408	8531	12,696	18,556	18,040	20,214	23,325	29,438	41,599	103	225,910
1921 to 1930 ..	F.	43,178	7367	11,164	16,570	19,481	22,440	31,957	40,817	50,925	60	243,959
1931 to 1940 ..	F.	27,375	5965	9808	13,731	18,088	26,831	37,502	60,269	77,523	24	277,116
1941 to 1945 ..	F.	14,253	2321	3710	6040	7890	14,394	22,960	35,916	55,387	4	162,875

published in the official statistics for security reasons. The practices in Australia with regard to this type of death have been mentioned already (Lancaster and Willcocks, 1950). In brief, the soldiers who died abroad in the first World War have been regarded as having emigrated from Australia, whereas in the second World War deaths in the armed forces were not recorded in the official statistics. Thirdly, ambiguity may be caused by varying definitions of "still-birth" and of "live-birth".

The Lack of Uniformity in the Definitions of "Still-birth" and "Live-birth", and the Consequent Confusion in the Meaning of "Infantile Mortality".

When making international comparisons it is always wise to investigate the definitions of the terms "live-birth", "still-birth" and "infantile death". In Australia it may be said that any child showing signs of life, such as breathing or movements after separation from the body of his mother, will be registered and will appear in the official statistics as a "live-birth", although definitions vary from State to State. In some countries, including notably Belgium and the Netherlands, which have an infantile mortality rate of the same order of size as the Australian rate, children who die within the first three days before registration are registered as "presented dead". If a public holiday should intervene the period may be extended to six days. In these countries the statistics of the "presented dead" are given separately, and such births and deaths do not appear otherwise in the official statistics. In Italy, on the other hand, children who are born and die before registration are registered as "still-born", but for statistical purposes are counted as "live-births" and "infantile deaths". Signed articles on this topic and some of the implications of the divergencies of practice may be found in two numbers of the "Epidemiological Reports of the World Health Organization" (Pascua, 1948; Stowman, 1949). Even within a country, practices may vary because registration comes within State law and not Federal law.

An Anomaly in Definition of "Premature Births".

By means of a definition, there has been an attempt in Australia to limit the registration of still-births to fœtuses of over seven months' gestation, a fœtal length of 14 inches, or a fœtal weight of 44 ounces being used as a measure of maturity. A corresponding lower limit is given for "premature" births. Under the limits defined it is possible for a fœtus to be born as a "live-birth", but not as a "still-birth", in so far as the official statistics are concerned. Since the fall of infantile mortality rates throughout the civilized world, comparatively small differences will give a State or country the leadership in the infantile mortality rates. Increasing attention is therefore being given to the legitimacy of the exclusion from the mortality statistics of the very immature fœtuses delivered, for it is found that the fœtuses of weight under, say, 19 ounces have very slender chances of survival under present conditions, and yet the statistics treat them in the same tables as the usual run of infants born at term. This question is really full of difficulties. It is most difficult to determine at what age the fœtus becomes viable. The medico-legal text-books are singularly uninformative on the question. It appears essential, if all abortions are not to be registrable (as they are in the State of New York), that there should be a lower limit to the length of gestation at which still-births are notifiable. It seems more difficult to set the lower limit to the length of gestation of "live-births" for registration purposes. Probably we shall not err if we continue to register as a "live-birth" all infants, whatever their presumed length of gestation, who show any signs of life, for we can easily imagine advances in technique that would give any product of conception born alive a good chance of survival.

TABLE II.

The Measurement of Mortality in Australia.

The Deaths in Australia in Childhood by Sex and by Single Years of Life, and by Certain Other Age Groups Not Already Detailed in Table I for Five Periods.

Period.	Sex.	Deaths in the Age Groups (Years).											
		0.	1.	2.	3.	4.	1 to 4.	5 to 9.	10 to 14.	15 to 19.	20 to 24.	0 to 14.	15 to 44.	45 to 74.
1908 to 1910 ..	M.	14,405	2378	899	566	422	4265	1394	1118	1853	2291	21,182	15,298	28,685
1911 to 1920 ..	M.	50,156	9155	3814	2459	1805	17,233	5872	4013	5912	8462	77,274	59,659	118,053
1921 to 1930 ..	M.	41,918	7170	3006	2100	1567	13,843	5323	4163	5948	7234	65,247	53,755	144,524
1931 to 1940 ..	M.	26,638	3951	2084	1549	1203	8787	4606	3893	5718	7216	43,924	48,856	178,015
1941 to 1945 ..	M.	14,603	1827	944	629	554	3954	1853	1617	2266	2083	22,027	18,459	101,938
1908 to 1910 ..	F.	11,172	2036	828	510	388	3762	1241	930	1629	2224	17,105	13,369	17,925
1911 to 1920 ..	F.	38,297	7832	3323	2195	1761	15,111	5193	3338	4848	7848	61,939	49,292	72,977
1921 to 1930 ..	F.	31,676	5949	2504	1728	1321	11,502	4260	3107	4468	6696	50,545	47,215	95,214
1931 to 1940 ..	F.	20,007	3368	1748	1212	1040	7368	3379	2586	3955	5853	33,340	41,627	124,602
1941 to 1945 ..	F.	11,077	1502	710	537	427	3176	1288	1033	1483	2227	16,574	17,040	73,270

TABLE III.[1]

The Measurement of Mortality in Australia.

The Years of Life Experienced by Individuals in Age Groups.

Period.	Sex.	Years of Life (in Thousands) Experienced by Persons in Age Groups by Sex: Age Groups in Years.									
		0 to 4.	5 to 14.	15 to 24.	25 to 34.	35 to 44.	45 to 54.	55 to 64.	65 to 74.	75 and Upwards.	Total.
1908 to 1910	M.	775	1269	1365	1074	852	701	334·5	196·7	90·61	6659
1911 to 1920	M.	2848	5016	4595	4106	3295	2601	1611	786·7	322·82	25,181
1921 to 1930	M.	3152	6075	5341	4756	4241	3060	2288	1213·1	401·51	30,527
1931 to 1940	M.	2849	6212	6163	5484	4674	3991	2643	1636·6	623·59	34,275
1941 to 1945	M.	1638	2826	3153	2949	2549	2176	1628	880·5	394·18	18,194
1908 to 1910	F.	759	1259	1313	1001	760	554	276·8	178·6	79·12	6181
1911 to 1920	F.	2796	4985	4598	4057	3088	2250	1383	718·6	321·01	24,196
1921 to 1930	F.	3049	5935	5132	4728	4041	2855	2078	1099·9	433·97	29,350
1931 to 1940	F.	2737	6004	5972	5167	4667	3863	2598	1653·4	688·14	33,351
1941 to 1945	F.	1573	2724	3070	2901	2433	2228	1636	963·9	473·77	18,003

[1] The various populations at risk for the two periods, 1908 to 1910 and 1911 to 1920, have been obtained by summation from a table of M. J. Holmes (1925), the data for which were evidently supplied by the Bureau of Census and Statistics, Canberra, although no confirmation of this can now be obtained, except that the annual totals agree exactly with those of the final revised intercensal estimates for the total population. The estimates for the years 1921 to 1940 were kindly supplied by the Bureau to me and are to be regarded as final up to the census year 1933. The estimates for the years since 1933 and so for the years 1931 to 1940 are to be regarded as provisional estimates only. The estimates for the years 1941 to 1945 have been derived from the intercensal estimates for 1941 and 1945 of *Demography*, on the assumption that the population in each age group increased in arithmetical progression over the years 1941 to 1945 and are also provisional.

Suggestions to the contrary have been made, some at least of which are inconsistent with the proposer's own experience. Thus B. Meredith (1950), in an otherwise excellent paper on infantile mortality in Victoria, suggests that children under the weight of 44 ounces should be classified as "non-viable", whereas in her Table XIII it is shown that at the Women's Hospital, Melbourne, out of ten infants with a birth weight under 32 ounces one survived, and of 11 infants with a birth weight between 32 ounces and 40 ounces, no less than eight survived early infancy.

Comment.

It appears, then, that we must continue to register and to include in the statistics of infant births all live births regardless of whether the fœtuses are stated to be "non-viable" or "pre-viable". Any deaths in this class will then automatically come under the heading of "infantile death". It is important to be able to determine, however, how many infant deaths are due to this excessive degree of immaturity, more especially since the bulk of the infant deaths nowadays are ascribable to immaturity. One possible solution is to provide special forms for infant deaths on which some of the more relevant details could be given. Foremost among these details are age, parity and disease of mother, weight of infant and plurality of birth. It is to be regretted that owing to the registration system the mortality of multiple births is not shown separately from that of single births in the official statistics in Australia generally. Of course, an alternative to the provision of new forms is a standing order that a representative of the health department in each State should obtain these data. Since the vast majority of confinements in Australia occurs in hospital, the collection of this material would not be difficult. We suggest finally that, regardless of how the information is obtained, there should be a tabulation additional to the usual infant mortality tables showing the birth weight where it is given on the certificate of infants that die, and stating whether they have been labelled as "non-viable" and "pre-viable". It would be desirable to have the corresponding information for all confinements.

DEATHS FROM ALL CAUSES IN AUSTRALIA.

The deaths for all Australia are given in *Demography*, an annual bulletin of the Bureau of Census and Statistics, Canberra, by age and sex and by cause of death in the main tables, with the International List as a guide to classification. The ages are always to be read as "age last birth day". The deaths are detailed by single years for the first five years of life and then are given by five-yearly age groups up to one hundred years. Additional groups are "over one hundred years" and "age unstated". In other tables, certain of the mortality data are treated with the aid of alternative age groups more appropriate to the particular purpose; for example, the total deaths and the

TABLE IV.

The Measurement of Mortality in Australia.

The Years of Life Experienced for Certain Periods by Children in the First Five Years of Life by Single Years of Life and by Children and Young Adults in the Five-yearly Age Groups in the Ages Five to Twenty-four Years Last Birthday.

Period.	Sex.	Hundreds of Years of Life Experienced by Persons in the Age Groups (Years).								
		0.[1]	1.	2.	3.	4.	1 to 4.	5 to 9.	10 to 14.	15 to 19.	20 to 24.
1908 to 1910	M.	1758	1547	1498	1460	1410	5916	6538	6155	6793	6859
1911 to 1920	M.	6717	6053	5925	5807	5676	23,461	26,317	23,839	23,355	22,596
1921 to 1930	M.	6873	6398	6364	6249	6208	25,219	31,067	29,683	27,726	25,684
1931 to 1940	M.	5977	5709	5717	5756	5806	22,988	30,311	31,804	31,336	30,298
1941 to 1945	M.	3763	3352	3191	3069	2973	12,585	13,857	14,404	15,883	15,649
1908 to 1910	F.	1639	1464	1416	1373	1382	5636	6475	6114	6644	6487
1911 to 1920	F.	6380	5847	5739	5625	5498	22,709	26,104	23,743	23,095	22,881
1921 to 1930	F.	6512	6164	6077	6025	5991	24,257	30,192	29,154	26,825	24,491
1931 to 1940	F.	5686	5481	5490	5524	5570	22,065	29,238	30,804	30,405	29,319
1941 to 1945	F.	3581	3224	3066	2949	2862	12,101	13,351	13,888	15,373	15,329

[1] These are the number of births, not the number of years lived in the period. The years of life experienced at the ages one, two, three and four years have been computed as explained in the text from the statistics of births and of deaths at these ages, migration being neglected.

TABLE V.

The Measurement of Mortality in Australia.

The Birthplaces of Children under Five Years of Age at Successive Censuses in Australia.[1]

Date of Census.	Males.			Females.		
	Recorded as Born in Australia.	Recorded as Born Outside Australia.	Total Males at Ages 0 to 4 Years.	Recorded as Born in Australia.	Recorded as Born Outside Australia.	Total Females at Ages 0 to 4 Years.
1911	263,352	2920	267,411	256,096	2754	258,222
1921	301,348	3775	305,522	290,653	3607	294,684
1933	289,857	604	290,461	277,836	668	278,504

[1] The numbers for 1911 and 1921 evidently include some with birthplaces unrecorded, so that the totals do not agree with the total recorded figures.

deaths by the main causes are given in fine age grouping for the first year of life.

The total deaths over the years of the periods under discussion have been summed, and are shown in Table I by age and sex. The age groups are those commonly used in standardization, commencing with an age group of the first five years and then seven ten-year age groups with a group "seventy-five years and over" and a group of unstated ages. For the study of some diseases it is convenient to have a somewhat finer subdivision at the earlier ages, so in Table II these deaths are shown by single years of age for the first five years, and then the deaths are given up to the age of twenty-four years by five-yearly age groups.

It is frequently of interest to know what proportion of deaths are due to individual causes. It is unnecessary usually to compute this proportion for every age group, but at the same time it may be misleading to give such a proportion by using the deaths at all ages combined. Such ratios have been used in the case of cancer deaths and have led to false conclusions, since the age distributions of the populations compared may differ greatly. A compromise is effected by taking the proportion in fairly wide age groups, such as nil to fourteen years, fifteen to forty-four years, forty-five to seventy-four years, seventy-five years and upwards. In Table II are supplied the deaths in the required age groups which are not already given in Table I.

Unstated Ages at Death.

In the years 1908 to 1910, among the male deaths some 0·26% of the ages and among the female deaths some 0·06% of the ages were unstated. These percentages of unstated ages have steadily diminished, until in the period 1941 to 1945 only 0·018% of the male and 0·002% of the female deaths were recorded with age unstated. A similar problem arises at the time of the censuses. In this case the unstated ages are distributed between the age groups according to arbitrary rules. Since the proportion is so small in the case of deaths, I have ignored these deaths at unstated ages in the computation of the age-specific rates, more especially as I believe that these unstated ages are usually those of old men, and that unstated ages are likely to be associated with vague or unstated causes of death. Furthermore, any distribution of the deaths at unstated ages to the various age groups by arbitrary rules would have led to further computation and to other difficulties in application, especially in diseases restricted to narrow ranges of the life span.

The Population at Risk.

In a previous paper, we (Lancaster and Willcocks, 1950) have briefly reviewed the practices regarding the enumeration of the population at the time of the census and the formation of the intercensal estimates. From these, estimates of the population at risk have been constructed. For a single calendar year the concept of the population "at risk" presents no difficulty; thus if the death rate for the whole population is to be computed for a certain year, then we should take the population at the mid-year as the measure of the mean number of persons alive throughout the year, and so of the number of years lived by persons at risk in the year. If the deaths, pooled over a number of years, are being considered, then we should calculate the number of persons at risk in the following manner. In the first year there will be, say, "P_1" persons alive at the mid-year, in the second year there will be "P_2" persons and so on up to the tenth year when there will be "P_{10}"

TABLE VI.

The Measurement of Mortality in Australia.

The Age-Specific Mortality Rates Expressed as a Rate per Million per Annum.[1]

Period.	Sex.	Death Rates per Annum per Million of Persons Exposed to Risk at Ages (Years).								
		0 to 4.	5 to 14.	15 to 24.	25 to 34.	35 to 44.	45 to 54.	55 to 64.	65 to 74.	75 and Upwards.
1881 to 1890	M.	45,494	3221	6637	8788	11,287	18,223	32,176	63,169	144,724
1891 to 1900	M.	37,775	2666	4540	6910	9587	15,045	29,916	59,764	146,646
1901 to 1910	M.	28,007	2073	3539	5109	8097	13,547	25,319	58,805	149,640
(1908 to 1910)	M.	24,087	1989	3036	4404	7540	12,698	25,191	57,138	142,677
1911 to 1920	M.	23,662	1971	3128	5050	7450	13,346	25,465	53,796	153,519
1921 to 1930	M.	17,691	1562	2468	3509	5632	11,009	22,443	49,042	134,684
1931 to 1940	M.	12,485	1368	2099	2657	4569	9624	21,861	50,004	132,434
1941 to 1945	M.	11,331	1228	1379	1720	3545	9134	22,005	52,521	140,406
1881 to 1890	F.	40,349	2979	4990	7952	10,281	13,997	23,291	51,501	131,609
1891 to 1900	F.	32,759	2450	3802	6382	8421	11,244	21,893	45,192	128,813
1901 to 1910	F.	23,648	1866	3201	5010	7072	9581	18,528	45,936	124,417
(1908 to 1910)	F.	19,676	1725	2934	4616	6440	8975	18,136	44,400	119,489
1911 to 1920	F.	19,104	1711	2761	4574	5843	8983	16,866	40,964	129,588
1921 to 1930	F.	14,161	1241	2176	3505	4821	7861	15,379	37,110	117,348
1931 to 1940	F.	10,002	993	1642	2657	3875	6946	14,435	36,451	112,655
1941 to 1945	F.	9059	852	1208	2082	3243	6462	14,030	37,262	116,907

[1] These rates were computed from estimates of the populations at risk before they were rounded off so as to be suitable for inclusion in Tables III and IV. It will be found that the rounded-off divisors, when used, yield rates differing from the above figures and those of Table VII only by rounding off errors in the final digit in most cases. In every case the proportionate error is trivial.

persons alive at the mid-year. Then we find that "P_1" years of life have been experienced by the population in the first year, "P_2" in the second year, and so on. So that the total number of years experienced in the ten-year period would be $(P_1 + P_2 + \ldots + P_{10})$ years. To obtain the mortality rate for the ten years, the deaths over the ten years are summed, and the sum so obtained is divided by the total number of years experienced in the corresponding period. In Table III are shown the number of years experienced in the age groups that will be used for standardization and so corresponding to the deaths of Table I. In Table IV the populations at risk at the earlier ages are given in more detail—firstly the number of births in the period, which is the divisor for the infant mortality rates, and then the populations at risk for single years of life up to age four years last birthday, and finally the populations for the four five-yearly age groups up to the age of twenty-four years last birthday.

Computation of the Populations at Risk in the First Five Years of Life.

The populations for the first five years of life have been computed as follows. Approximately half the years lived at age under one year in 1941 will be lived by children born in 1941, half will be lived by those born in 1940. But of these not all will live a complete year, so that a correction is made equal to half the mean of the deaths at ages under one year in the years 1940 and 1941. Similarly the years of life lived by children aged one year in 1942 will be obtained as the mean of the births in 1940 and 1941 less the mean of the deaths at age nil in 1940 and 1941 and less half of the mean of the deaths at the age of one year in 1941 and 1942. What, in effect, we are doing is to estimate the years lived in the various calendar years by a cohort or group of children born, on an average, at the beginning of 1941. By a repetition of this process we have built up the estimates of the child populations for the Commonwealth over the five periods mentioned earlier. Errors would be introduced by migration; but it appears that these are not likely to be large, especially in the earliest years of life. Table V has been formed from the tables in the statistician's reports on the censuses of 1921 and 1933. We find that the number of children under the age of five years born overseas was of the order of 1% of the total child population at the corresponding age, and hence that the error introduced by migration is relatively unimportant.

For the mortality of the first year of life the divisor in computing mortality rates is not the children at risk but the number of births for the period of time, giving the "infant mortality", which corresponds to q_0 of the actuary.

The Sex-Specific and Age-Specific Death Rates.

For any serious comparisons of mortality rates it is necessary to compare the death rates of classes of persons similar particularly with regard to age and sex. Since, in general, we shall be treating the males and females as belonging to two separate populations, we shall use the term "age-specific" as synonymous with "sex-specific and age-specific". It has been found by general experience that for most purposes the age groups as set out in Table I are the most satisfactory, since there is some need to limit the number of operations to be performed while retaining age groups that are comparable throughout a great range of likely populations, for wider age groups, say fifteen to forty-five years, may not be strictly comparable between rapidly growing and declining or stationary populations. Differences in the age distribution within the ten-year age groups between different populations have generally been found to be trivial.

The Definition of the Age-Specific Death Rates.

The term "specific" death rate may be used in connexion either with deaths due to a particular cause or with deaths of a certain class of individuals specified by age or by sex or occupation or other attribute. The age-specific mortality rates are obtained by dividing the number of deaths occurring in a certain age group in a period of time by the number of years of life experienced by persons of the same sex and age group in that period. The death rates are usually given as deaths *per annum* per thousand, million, or other power of ten, of persons "at risk" or years of life experienced. I shall give the rates as rates per million as a rule. For a single year the computation of the age-specific death rates presents no difficulty. We find, for example, that in 1933 there were 1203 deaths of males aged fifteen to twenty-four years last birthday. The population of the corresponding age group in Australia as enumerated at the census of April 30, 1933, and subsequently "adjusted", was 609,793. We take this as the mean population throughout the year. So the age-specific death rate of males aged fifteen to twenty-four years is $1{,}000{,}000 \times \frac{1203}{609{,}793}$ per million *per annum*—that is, 1973 per million *per annum*. If the experience over a period—say for the years 1931 to 1940—is considered, then the appropriate formula is a slight extension of that above, namely:

$$m = \frac{\text{sum of deaths for each year 1931 to 1940}}{\text{sum of mean populations for each year 1931 to 1940}} \quad (1)$$

where m is the age-specific mortality for males aged fifteen to twenty-four years and the deaths and populations used refer to this same age group. The deaths were, in fact, 12,934, and the number of years experienced, or the sum of the mean populations for each year, was 6,163,381. So

TABLE VII.

The Measurement of Mortality in Australia.

The Death Rates in Childhood and Young Adult Life from All Causes in Australia for Certain Periods.

Period.	Sex.	The Death Rates per Annum per Million of Persons Aged (Years).									
		0.[1]	1.	2.	3.	4.	1 to 4.	5 to 9.	10 to 14.	15 to 19.	20 to 24.
1901 to 1910[2]	M.	101,120	18,040	6770	4410	3500	—	2221	1915	3003	4095
(1908 to 1910)	M.	81,939	15,368	6001	3877	2993	7209	2132	1816	2728	3946
1911 to 1920	M.	74,676	15,125	6437	4235	3180	7345	2231	1683	2531	3745
1921 to 1930	M.	60,990	11,206	4724	3360	2524	5489	1713	1403	2145	2817
1931 to 1940	M.	44,565	6921	3645	2691	2072	3822	1520	1224	1825	2382
1941 to 1945	M.	38,803	5450	2958	2050	1863	3142	1337	1123	1427	1931
1901 to 1910[2]	F.	83,490	16,840	6310	4120	3250	—	2015	1709	2723	3697
(1908 to 1910)	F.	68,172	13,903	5848	3714	2807	6675	1917	1521	2452	3428
1911 to 1920	F.	60,024	13,395	5791	3902	3203	6654	1989	1406	2099	3430
1921 to 1930	F.	48,645	9651	4121	2868	2205	4742	1411	1066	1666	2734
1931 to 1940	F.	35,184	6145	3184	2194	1867	3339	1156	839	1301	1996
1941 to 1945	F.	30,933	4659	2316	1821	1492	2625	965	744	965	1453

[1] The infant mortality rate (see text).

[2] For 1901 to 1910, the rates have been taken from G. H. Knibbs (1917), "Census of the Commonwealth of Australia", Appendix A, Volume I, page 375.

that $m = 12{,}934 \times \frac{1{,}000{,}000}{6{,}163{,}381}$, or 2099, deaths *per annum* per million. Other specific rates may be defined either by classification of the persons at risk or by specifying particular causes or groups of causes of death. The only specialized type that is of immediate interest to us here is the sex-specific and age-specific mortality by cause of death.

The Use of Rates per Million.

We use rates per million *per annum* to avoid the decimal point, so troublesome in printing (a point especially mentioned by Udny Yule, 1934). Further, we tend to think of a human population in terms of millions rather than in terms of tens or hundreds of thousands. In some offices it is customary to give the rates as per ten thousand, but to give them to two decimal places. Our practice is quite equivalent to this, but avoids the decimals. The final figure in some cases cannot be defended from the point of view of sampling errors, but is retained in order to facilitate checking and to avoid the introduction of further error due to rounding off in standardization and other operations.

The Standard Error of the Mortality Rates.

The standard error of the mortality rates is usually computed as follows. Let q be the probability of dying and p the probability of surviving in a population n of given age and sex; then the standard error of q is given by

$$\text{S.E. } (q) = \sqrt{\left(\frac{pq}{n}\right)}, \quad \ldots\ldots\ldots\ldots\ldots\ldots (2)$$

the observed p and q being used as an approximation to the true values. But q is small, as a rule, and p near to unity, so that

$$\text{S.E. } (q) = \sqrt{\left(\frac{q}{n}\right)}, \text{ nearly } = \frac{1}{n}\sqrt{(nq)} \quad \ldots\ldots\ldots\ldots (3)$$

So the standard error of the observed q may be taken as $\sqrt{\frac{\text{(number of deaths)}}{n}}$.

If the rate is given per million

$$\begin{aligned}\text{S.E. (rate)} &= 1{,}000{,}000 \sqrt{\left(\frac{q}{n}\right)} \\ &= \frac{1{,}000{,}000}{n} \sqrt{\text{(number of deaths)}} \\ &= \frac{\text{(rate per million)}}{\sqrt{\text{(number of deaths)}}} \quad \ldots\ldots (4)\end{aligned}$$

Alternatively, the sampling variance of the number of deaths is npq, which is approximately nq, since p is nearly unity. The sampling variance of the rate is then given as follows:

$$\text{Sampling variance (rate per million)} = nq \times \left(\frac{1{,}000{,}000}{n}\right)^2 \quad \ldots\ldots (5)$$

which suggests an easy method of obtaining the variance of a standardized rate.

An example will illustrate the method. Let us suppose that there were d deaths in an age group containing n individuals, and that there are s individuals in the corresponding age group of the standard million. The expected deaths in the age group of the standard million will be given by $\frac{ds}{n}$, the sampling variance for this expected number will be $\frac{ds^2}{n^2}$. We may use the theorem that the variance of a sum is the sum of the variances to obtain:

The variance of the expected deaths in a standard million $= \Sigma \frac{ds^2}{n^2}$, where summation is to be performed over every age group. ..... (6)

A Note on the Age-Specific Mortality Rates and the q_x.

The measurement of mortality has been discussed for various reasons more by actuaries than by medical or health workers. The actuary has always the concept of a life table before him and the need for the computation of assurance premiums and annuities, so that mortality surveys have always tended to be expressed in actuarial terms, q_x being used to describe mortality; q_x is defined as the probability (or relative frequency) of a person, aged x years exactly, dying before he or she attains the age of $(x + 1)$ years. In actuarial notation, the age-specific rates in the population are written as m_x. Here m_x is equal by definition to the number of deaths of persons aged x last birthday in a year divided by the mean number of persons living during the same calendar year. m_x and q_x are easily found to be related by the formula:

$$q_x = \frac{2m_x}{2 + m_x}, \quad \ldots\ldots\ldots\ldots\ldots (7)$$

$$m_x = \frac{2q_x}{2 - q_x}, \quad \ldots\ldots\ldots\ldots\ldots (8)$$

after it has been assumed that deaths occur at a constant rate throughout the year in persons passing from age x years to $(x + 1)$ years exactly.

When they are small, the q_x and m_x approximate closely. The q_x have been regarded after "smoothing" as describing the mortality better than the m_x, and so better than the observations themselves, for in the computation of the q_x processes of smoothing are used that rid the data of many of the irregularities which are due sometimes to sampling errors and sometimes to natural irregularities, such as the diminished rate of increase in the mortality rates in the male in the twenties. However, let us quote the noted actuary Sir William Elderton in his discussion on the paper of Pedoe (1947), expressing a growing body of opinion thus:

> To proceed from the death rate (i.e. the ratio of deaths to population—which was what Farr had in mind) to q_x involved making a calculation and getting away from a fact to a theory based on assumptions that were interwoven with the idea of a stationary life table population of the l_x type.

In the data that are available to the actuary, the ratios q_x are often to be directly observed; for example, the actuary may be studying a group of policy holders followed from entry until their exit from his field of interest. The number of survivors to each age will be the l_x, and the q_x and p_x are quite simply derived. Data are rarely available to the vital statistician in this form except sometimes in the follow-up of patients post-operatively, and in the follow-up of tuberculosis or diabetes clinics.

The Reasons for the Adoption of the m_x rather than q_x as a Measure of Mortality.

The use of m_x has several advantages over the q_x. Firstly, the q_x require as a rule one further computation than the m_x. Secondly, the m_x are more readily partitioned into components due to individual causes of death. Thirdly, in the age-groups above seventy-five years the q_x obtained from the life tables are often greatly affected by interpolation methods. On the other hand the q_x may often be available for comparison when the m_x are not, as in the various reports on the census in Australia. However, it is well to note here the very detailed description of the data for the life tables formed in conjunction with the census of 1911 (G. H. Knibbs, 1917). In the reports on later censuses this valuable section has not been repeated, from reasons of economy, it would appear.

The Age-Specific Mortality Rates in Australia from All Causes.

In Table VI are shown the age-specific rates of mortality (all causes) for the five periods, and also are given the corresponding mortality rates for Australia for the three decennia 1881 to 1910 computed from tables in the statistician's report on the census of 1911. In Table VII there

is a more detailed analysis for the mortality under twenty-five years of age.

In the discussion on tuberculosis mortality (Lancaster, 1950*a*), the three methods of reading such a table have been explained in some detail. By the first method it may be read by rows to obtain the mortality rates for a given period. This is exemplified by Figure I, in which the mortality rates for the years 1931 to 1940 are displayed. Certain features are immediately apparent. In the first five years of life the mortality is high (in Figure I and Table VI the infantile deaths make the chief contribution to the mortality of the under five years age group). The mortality rate attains a minimum in the age group five to fourteen years and then steadily rises throughout life. Our groupings are too coarse to bring out the finer points of the mortality, but we may use the mortality of the three years about the 1933 census to compute the mortality by single years of life. It is then found that there is a minimum for the rates in both sexes at age eleven years. The rates increase from this age up to the age of twenty-four years, but then the rate of increase becomes less and the rates remain practically stationary up to the age of thirty years or so. This feature is especially pronounced in the case of the males. In some countries of the world there is actually a decline of the rates in the late twenties —that is, a relative maximum occurs in the rates about the age of twenty-four years. When this has been investigated in other countries, common explanations have been violence in the males, the hazards of child-bearing in the females and tuberculosis in both sexes. Under modern conditions the maximum, or tendency to it, is usually due to violence. Figure I is drawn on a semi-logarithmic scale and brings out a point of some interest, that the logarithm of the death rates can be satisfactorily represented by a straight line over a considerable proportion of the life span. This is the Gompertz law—that the mortality rates increase in geometrical progression with increase of age, or in other words, that the logarithm of the mortality rates increases according to a linear relation with the age. A review of the meaning, if any, that can be attached to the constants of the straight line has been written by Greenwood (1928). It is found that the Gompertz formula or Makeham's modification of it does summarize the mortality experience from the age of ten years onwards in a remarkably efficient manner. Treatment of Australian data by these formulæ will be found in a paper of A. H. Pollard (1949).

The second method of studying the death rates is by following the mortality of a given age group over the different periods. This is to read Table VI or Table VII by columns. Such a comparison has been shown graphically in Figure II, in which the scale is again semi-logarithmic. Its slope is thus a measure of the relative rate of decrease of the rates. It is then found that the greatest relative improvement in the death rates has been at the earlier ages and that there has been little improvement at the higher ages. Figures II and III are drawn to the same vertical and horizontal scales.

The third method of study of the rates is the generation method, which is explained in greater detail in the paper on tuberculosis mortality. In the study of tuberculosis mortality rates the method leads to an important reinterpretation of the shape of the curve of the mortality rates at a point of time.

The Masculinity of the Death Rates.

The masculinity of the death rates for a given period is defined as 100 × male mortality rates divided by the female mortality rate of the same age group. Table VIII has been constructed by the use of this formula. A remarkable constancy may be noted at the early ages 0 to four years, although very great changes have occurred in the level of the mortality rates at these ages. At the highest ages the masculinity of the rates has also been almost constant. The effect is less pronounced in the intermediate age groups, but nowhere is the variation in the ratio to be considered large. The more favourable death rates of the females have been commented on many times (see for example F. A. E. Crew, 1948). This superiority of the

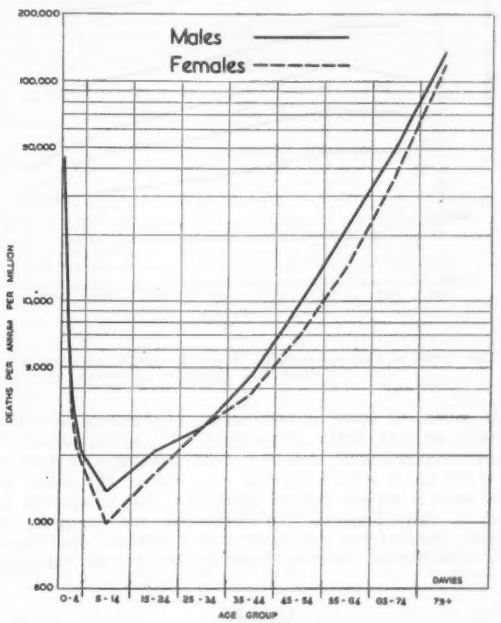

FIGURE I.

The death rates in Australia from all causes by age and sex for the decennium 1931-1940. Semi-logarithmic grid.

TABLE VIII.

The Measurement of Mortality in Australia.

The Masculinity of the Death Rates at Different Ages.[1]

Period.	Masculinity at Ages (Years).								
	0 to 4.	5 to 14.	15 to 24.	25 to 34.	35 to 44.	45 to 54.	55 to 64.	65 to 74.	75 and Upwards.
1908 to 1910	122	115	103	95	117	141	139	130	119
1911 to 1920	124	115	113	(110)	(128)	149	151	132	118
1921 to 1930	125	126	113	100	117	140	146	132	115
1931 to 1940	124	138	128	100	118	139	151	137	118
1941 to 1945	125	144	114	(83)	(109)	141	157	141	120

[1] The "masculinity" of the death rates has been computed by means of the formula: masculinity = 100 × male mortality rate divided by the female mortality rate. The ratio of male to female mortality, in other words, has been multiplied by 100 to obtain the masculinity as defined above. The two values for the years 1941 to 1945, which have been distinguished by parentheses, are probably spurious, as the deaths at these ages of males if they occurred in the armed forces would not appear in the official statistics, although the members of the armed forces are not shown as removed from the populations at risk. The two values for the years 1911 to 1920, distinguished by inclusion in the parentheses, are possibly spuriously high because of the recording of the fit military personnel, who departed overseas, as being emigrants. The remainder of the male population at military ages will, therefore, have been unfavourably selected.

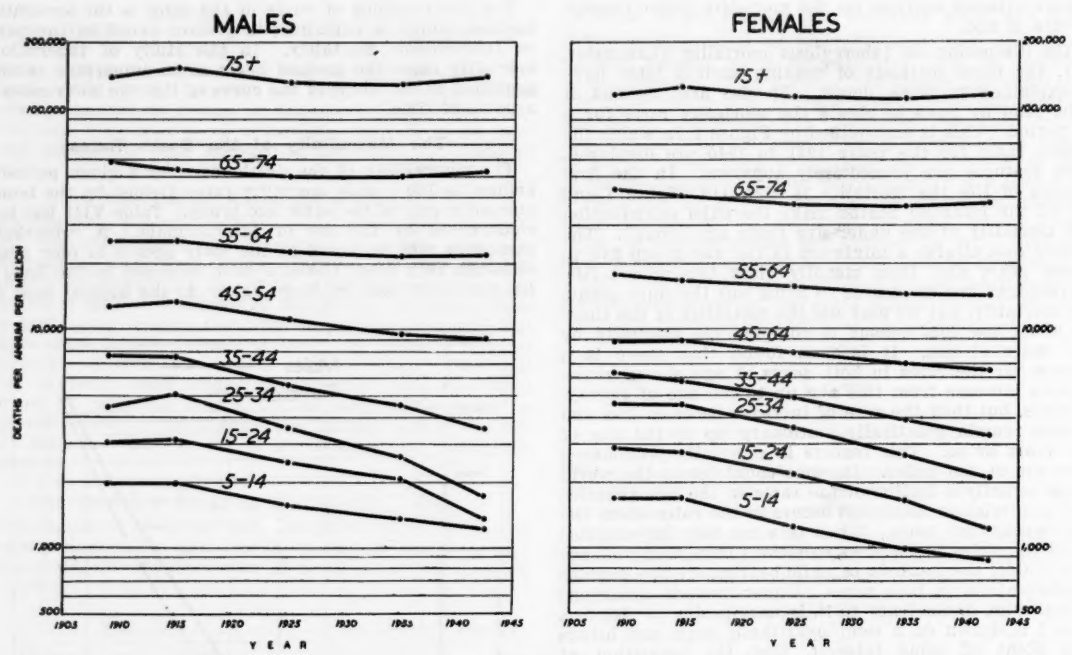

FIGURE II.

The trend of the age-specific and sex-specific mortality rates over the period 1908 to 1945. The age groups are indicated by the ages last birthday in years, written on the appropriate graph. Semilogarithmic grid.

female rests on some physiological mechanism of which we have as yet little knowledge. In some cases—for example, occupational risk and child-bearing—we can see that there are *a priori* reasons why one or the other sex should have a higher rate of mortality; but in general the reason is quite obscure. In childhood, for example, the mortality rates from diarrhœa and dysentery are higher in the male, but on the other hand mortality from whooping cough is higher in the female. Such sex differences defy explanation at the present time.

The Mortality Rates of the Groups Seventy-Five Years and Upwards.

Some doubt exists as to whether the death rates of the age group seventy-five years and upwards are comparable from period to period. The mortality at a given time is

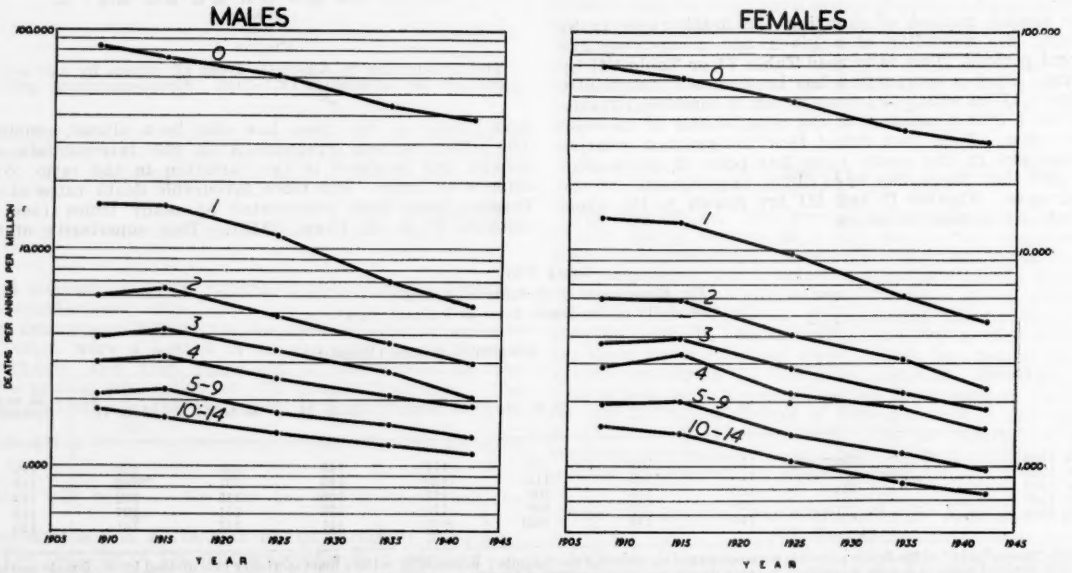

FIGURE III.

The trend of the age-specific and sex-specific mortality rates over the period 1908 to 1945. The age groups are indicated by the ages last birthday in years, written on the appropriate graph. The graphs marked "0" are the graphs of the infant mortality rates. Semi-logarithmic grid.

changing rapidly with advancing age at these high ages, and so the age group is too broad for any special investigation of mortality at these ages. It is possible that, in Australia, the age distribution in this group has changed considerably over the period of years considered. There are now relatively more of the very old than at the turn of the century. In mortality comparisons, then, the mortality rates for the later periods will be too heavily weighted with the mortality of the very old. In vital statistical work it is often felt that the further analysis of mortality in this age group presents great difficulty, although a great body of data is now available from assurance experience.

TABLE IX.

The Measurement of Mortality in Australia.

The Trend of the Mortality in Australia as Measured by the Crude and Standardized Death Rates.

Period.	Sex.	Death Rates per Thousand per Annum.			
		Crude Death Rate.	As Standardized onto the Enumerated Population of England and Wales, 1901.	As Standardized onto the Life Table Populations Derived from the Australian Census of 1933.	As an Equivalent Average Death Rate for Persons Under 65 Years of Age.
1908 to 1910	M.	11·7	11·5	19·0	10·3
1911 to 1920	M.	12·1	11·6	19·3	10·5
1921 to 1930	M.	10·4	9·5	16·5	8·5
1931 to 1940	M.	10·3	8·3	15·5	7·5
1941 to 1945	M.	10·9	7·9	15·5	6·9
1908 to 1910	F.	9·4	10·0	17·5	8·1
1911 to 1920	F.	9·3	9·8	17·5	7·7
1921 to 1930	F.	8·3	8·2	15·3	6·5
1931 to 1940	F.	8·3	7·1	14·1	5·5
1941 to 1945	F.	9·1	6·7	14·0	5·0

Standardized Rates of Mortality.

It is necessary to stress that the most satisfactory comparison of the mortality of two populations is that by the age-specific rates. This involves the comparison of some nine pairs of numbers, so it is usually thought desirable to have a single measure of mortality which will summarize the mortality and yet be independent of the age distributions of the populations compared. Standardization is one such method. In essence, standardization determines the number of deaths that would occur in a standard population if it were exposed at every age group to the same mortality risks as obtained in the observed population. The arithmetic of the process is illustrated in the paper on the cancer rates in Australia (Lancaster, 1950*b*). There are several types of population to be used for the standard. An actual population enumerated at a census may be used, commonly in English-speaking countries the standard million, derived from the census of 1901 in England and Wales. The great defect in this population is the small proportion of elderly persons, due to the previous era of high birth rates and rapid population growth in England and Wales. This resulted in the persons of seventy years of age being the survivors of a much smaller number of births than those aged twenty years and hence being "under-represented".

In a life table population, which may readily be constructed from the life tables, the death rate is the reciprocal of the expectation of life. This seems a natural conclusion to draw from any standardized rate. However, it is easily seen in Table IX, for example, that the reciprocal of the standardized death rates gives absurd values to the expectation of life when we standardize the mortality rates on to such an actual population as the population of England and Wales of 1901, obtaining a value of 141 years for the expectation of life at birth (that is $\frac{1000}{7{\cdot}1}$), which is clearly absurd. The error is that the only case in which the relation does hold is when the rates are standardized on to the life table population formed from the rates themselves. The absence of this relationship, once it is recognized, does not detract greatly from the value of the process of standardization, for it is then seen that standardization is merely a convenient system of weighting the rates at different ages, in order that comparisons may be made independent of the age distributions of the populations considered.

A good illustration of the value of standardization is given by Dublin and Lotka (1937). They found a good correlation between the "true death rate" or "life-table death rate" (that is, the rate in the life-table population or the reciprocal of the expectation of life) and the rates standardized on to the population of England and Wales, 1901; whereas the crude death rates gave a much lower correlation with the "true death rate". The populations compared were those of the 48 States of the United States of America.

Another simpler type of standard population was introduced by the office of the Registrar-General of England and Wales. In it every age from birth to sixty-five years is equally represented. This standard population, "the population of the equivalent average death rate", with a "rectangular" age distribution, is an approximation to the first sixty-five years of the life-table population, since deaths are relatively few between the ages of one and sixty-five years, and so the life-table population "pyramid" is practically rectangular for recent Australian data up to the age of sixty-five years. The corresponding rate is known as the "equivalent average death rate". It is particularly easy to compute—in fact, it is merely the average of the death rates of the first thirteen five-year age groups. If any of the death rates are given for a ten-yearly age group, they are counted twice, since the ten-year age group has a death rate approximately equal to half the sum of the two five-year age groups it contains. The equivalent average death rate has been much used for occupational mortality studies.

The Crude Death Rates.

The crude death rate is merely the ratio of the deaths *per annum* in a population to the mean number of persons alive in the population at any time. It has the great defect of being dependent on the age distribution of the populations considered. However, although the population of Australia has been aging, we note in Table IX that the crude death rate has been falling.

The Trend of the Mortality Rates in Australia.

From an examination of either Table VI or Figure II, it is apparent that there has been a great decline in the age-specific death rates generally in Australia. Ife we confine our attention to the female rates for the moment, we see that the fall was first evident between the decennia 1911 to 1920 and 1921 to 1930. The effect was originally greatest in the earliest age group, but now it appears to be most pronounced in the age group twenty-five to thirty-four years. In Table X such comparisons are facilitated by the giving of the rates as a percentage of those in the decennium 1901 to 1910, which has more claims to be considered normal than any other decennium in Australian history. Improvement in the age group seventy-five years and over is problematical and cannot be considered further without breaking down the age group. It is not possible at this stage to give a comprehensive statement on the decline of the Australian death rates. I propose to do so at a later date after reviewing the chief diseases and violence in detail.

The Forecasts of G. H. Knibbs.

In comparing the forecasts of Knibbs with the actual events, it is well to note the common experience in vital statistics that an unexpected turn of events may render eminently reasonable forecasts false. Knibbs (1917), in his report on the census of 1911, had at his disposal the

TABLE X.

The Measurement of Mortality in Australia.

The Mortality Rates in Each Age Group Expressed as a Percentage of the Corresponding Rate in the Years 1901 to 1910.

Period.	Sex.	Age Group (Years).								
		0 to 4.	5 to 14.	15 to 24.	25 to 34.	35 to 44.	45 to 54.	55 to 64.	65 to 74.	75 and Upwards.
1881 to 1890 ..	M.	162	155	188	172	139	135	127	107	97
1891 to 1900 ..	M.	135	129	128	135	118	111	118	102	98
1901 to 1910 ..	M.[1]	100	100	100	100	100	100	100	100	100
1911 to 1920 ..	M.	84	95	88	99	92	99	101	91	103
1921 to 1930 ..	M.	63	75	70	69	70	81	89	83	90
1931 to 1940 ..	M.	44	66	59	52	56	71	86	85	89
1941 to 1945 ..	M.	40	59	39[2]	34[2]	44[2]	67	87	89	94
1881 to 1890 ..	F.	171	160	156	159	145	146	126	112	106
1891 to 1900 ..	F.	139	131	119	127	119	117	118	98	104
1901 to 1910 ..	F.[1]	100	100	100	100	100	100	100	100	100
1911 to 1920 ..	F.	81	92	86	91	83	94	91	89	104
1921 to 1930 ..	F.	60	67	68	70	68	82	83	81	94
1931 to 1940 ..	F.	42	53	51	53	55	72	78	79	91
1941 to 1945 ..	F.	38	46	38	42	46	67	76	81	94

[1] The decennium 1901 to 1910, and perhaps the decennium 1921 to 1930, are the only two decennia in Australian demographic history that can be regarded as not abnormal, for they contained no war, no major depression, no gold rush of any great magnitude, and no wave of immigration.

[2] Statistical practices in war-time render these figures unsuitable for accurate comparisons.

mortality rates by age and sex for Australia for three decennial periods in the years 1881 to 1910. He fitted straight lines by the method of least squares (that is, the usual statistical procedure) to the logarithms of the mortality rates in successive decades and obtained the line of best fit. He then extrapolated by extending the straight line so obtained, and was led to forecast the rates for succeeding years. In Table XI are given his forecast rates for 1936, in order that they may be compared with the observed rates in 1931 to 1940. His forecast gives rates that are in general agreement with those observed but are rather too high in the age groups under five years. More recently, forecasts have been made by Pollard (1949).

TABLE XI.

The Measurement of Mortality in Australia: The Mortality Rates for 1936 in Australia as Forecast by G. H. Knibbs (1917).[1]

Age Groups. (Years.)	Forecast Rates of Mortality per Million per Annum for 1936.[2]	
	Males.	Females.
1	6000	5000
2	3000	4000
3	2000	3000
4	1000	1000
5 to 14	1400	1300
15 to 24	1800	1800
25 to 34	2600	2700
35 to 44	5300	3900
45 to 54	9700	6100
55 to 64	21,000	13,500
65 to 74	55,000	44,000

[1] "Census of the Commonwealth of Australia", Volume I, Appendix A, page 381.

[2] The rates are given here per million *per annum* to render them more readily comparable with the rates of Table VI for the years 1931 to 1940, with which they are evidently in quite good agreement.

SUMMARY.

A general survey of the methods of measuring mortality has been given, together with the data necessary for the computation of the various sex-specific and age-specific death rates. The definitions of a death have been briefly mentioned and the lack of uniformity of the definitions of a "livebirth" and a "stillbirth" has been noted. An anomaly as to the registration of births and stillbirths exists in Australia.

The deaths from all causes in Australia for five periods in the years 1908 to 1945 have been studied. These deaths are given in certain age groups, so that appropriate comparisons can be made when individual causes of death are studied. The treatment of deaths at unstated ages is noted.

The age-specific death rates have been defined. The advantages of using the age-specific mortalities rather than the q_x of the life tables have been mentioned. Standardization of the death rates has been explained.

Some general conclusions have been drawn. The trend of the death rates has been downwards. This effect has been most pronounced in the earlier years of life. Little improvement has occurred in the ages over sixty-five years, although the age groups selected are unsuitable for detailed comparisons over the age of seventy-five years. The superiority of the female rates over the male rates at all ages has been shown. Detailed discussion of the fall in the mortality rates must be deferred until the mortality rates from the more important diseases have been dealt with.

ACKNOWLEDGEMENTS.

I have to thank the Director-General of Health, Commonwealth of Australia, for permission to publish this article; Dr. A. H. Pollard, of the Mutual Life and Citizens Assurance Company, for helpful criticism; Mr. D. W. Davies, of this School, for drawing the diagrams; and Dr. Roland Wilson, Commonwealth Statistician, for permission to publish estimates of the populations at risk in the years 1921-1930 and 1931-1940, derived from the intercensal estimates 1921-1940, supplied by him to me.

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THE PROBLEM OF THE THORACIC STOMACH.[1]

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THIS communication is limited to a discussion of the thoracic stomach occurring as a complication of defects in the development of the œsophagus or the œsophageal hiatus of the diaphragm. Of Harrington's (1943) 295 patients surgically treated, 237 were in this group. It is a problem which interests physicians, surgeons, pædiatricians and radiologists.

Classification.

Akerlund's classification as quoted by Bockus (1946) is simple and satisfactory, and is as follows: (i) Congenitally short œsophagus producing a thoracic stomach. (ii) Para-œsophageal hiatus hernia. The lower part of the œsophagus remains in its normal position, and a portion of the cardiac end of the stomach herniates through the hiatus alongside the œsophagus. (iii) Œsophago-gastric hernia. Both the lower end of the œsophagus and a portion of the cardia prolapse through the hiatus.

Development.

In the congenitally short œsophagus some portion of the stomach is present in the thorax from birth. Symptoms may appear early but are often not prominent until middle life.

In the remaining types it is more than probable that a defect in the œsophageal hiatus exists from birth, but herniation does not occur until later in life, when it is due to some precipitating cause. Such a cause may be trauma producing a sudden rise in intraabdominal pressure. More commonly herniation is due to slowly increasing abdominal pressure from obesity or pregnancy, to which may be added a decrease in muscle tonus because of increasing age, and the use of tightly fitting corsets in an effort to overcome such defects and to mask the increasing obesity. For these reasons hiatus hernia and thoracic stomach occur most frequently in middle-aged females.

Mechanism of Symptoms.

The symptoms are complex and bizarre, but mainly arise from the following factors: (i) mechanical interference with the function of the herniated portion of the stomach; (ii) increased pressure in the posterior mediastinum; (iii) interference with the normal function of the diaphragm.

Compression and constriction of the herniated portion of the stomach may cause localized gastritis, chronic congestive changes and ulceration.

Increased pressure within the posterior mediastinum produces mechanical disturbance in the heart and sometimes the œsophagus. Palpitation, dyspnœa and cardiac pain may result. Œsophageal compression may cause intermittent dysphagia.

Diaphragmatic interference may add to the dyspnœa, and in one case seemed a factor in producing pulmonary atelectasis and subsequent bronchiectasis. It can cause left shoulder and arm pain.

A more serious effect may be seen in types (i) and (iii), in which the cardio-œsophageal junction is removed from the sphincteric action of the œsophageal hiatus. As a result gastric contents are permitted to be regurgitated much more readily into the œsophagus, causing œsophagitis and peptic ulcer of the gullet. Allison (1948) and Benedict and Sweet (1948) have stressed this complication and have emphasized the close association of peptic ulceration of the œsophagus in the presence of a thoracic stomach.

Symptoms.

Although this abnormality occurs most commonly in short, thick-set, middle-aged, multiparous females, it may present in subjects of either sex and at any age.

The most common complaints are of belching, epigastric fullness, substernal discomfort, nausea and vomiting and heartburn, symptoms which are frequent in many other gastro-intestinal disorders. However, the symptom complex most helpful in our series has been epigastric or low substernal pain radiating to the left hypochondrium and the left side of the back, aggravated by large meals, by reclining or by exertion.

The symptomatology may simulate many diseases. Peptic ulceration and cholecystic disease have been the commonest presenting diagnoses, but angina, coronary artery disease, stricture of the œsophagus, cancer of the cardia and intestinal obstruction have been provisional diagnoses. The simulation of coronary disease is particularly striking, and in those patients presenting with anginal symptoms in whom evidence of heart disease is minimal, the probability of hiatus hernia is considerable. Secondary anæmia with or without melæna and hæmatemesis is not unusual. As a cause of severe iron deficiency anæmia hiatus hernia is frequently overlooked, as has been emphasized recently by Schwartz and Blumenthal (1949).

In very young children there may be difficulty in swallowing, vomiting during and immediately after a meal, and even hæmatemesis.

Complications.

The condition would often remain unrecognized were it not for certain complications, such as the following.

1. Œsophagitis, œsophageal ulceration and stricture. The probable underlying mechanism has been explained. Burning substernal pain indicates this complication.

2. Gastritis and ulceration within the herniating portion of the stomach. Richard Bright is said to have described this complication in 1836.

3. Severe anæmia. Hæmatemesis and melæna may occur, but the hæmorrhage is most often of silent type, suspected only if the stools are examined for occult blood.

4. Malignant degeneration. Such changes may occur within the stomach, but it is unlikely that the incidence is higher because of this abnormality.

5. Perforation leading to mediastinitis or peritonitis.

6. Obstruction. Obstruction is uncommon in lesions associated with an hiatus hernia.

Concurrent Diseases and Abnormalities.

Congenital defects of the gastro-intestinal tract have been common in our series, particularly diverticula of the duodenum and colon. Cholelithiasis, cholecystitis, peptic ulceration and cardio-vascular disease have been frequent concurrent diseases.

Diagnosis.

Primarily the diagnosis is made or confirmed by radiological methods. Œsophagoscopy and gastroscopy are useful

[1] Read at a meeting of the Section of Medicine, Australasian Medical Congress (British Medical Association), Seventh Session, Brisbane, May-June, 1950.

adjuvants in determining the presence of œsophagitis, gastritis or ulceration, and in confirming or excluding malignant disease.

Large herniæ are easily apparent, but the smaller or reducible lesions are apt to escape detection unless the radiologist is constantly on guard.

Harrington (1940) has stressed certain points in the radiological diagnosis, as (*a*) displacement of the lower segment of the œsophagus, (*b*) a tortuous but not dilated terminal œsophageal segment, (*c*) an angulated segment, (*d*) undue retardation of the barium stream at the hiatus, (*e*) a level of the gastric contents above the œsophageal aperture, (*f*) apparent high hourglass contraction of the stomach.

At times in our series gastroscopic examination has been difficult because of deformity at the cardio-œsophageal junction obstructing the passage of the gastroscope. At other times it has been impossible to pass the instrument beyond the portion of the stomach within the thorax.

As the gastroscope is withdrawn, gastric mucosa can be seen in many cases to be present at 25 to 30 centimetres from the incisor teeth, whereas normally the cardio-œsophageal junction is seen at 38 to 40 centimetres.

Allison (1948) in describing the œsophagoscopic appearances, has stressed the presence of œsophagitis, ulceration and stricture. In those cases in which stricture does not exist, he has commented on the ease with which the œsophagoscope enters the stomach. This may be so in types (i) and (iii), but in the paraœsophageal type at times the cardio-œsophageal junction is beyond the vision of the œsophagoscopist.

Treatment.

Conservatism is essential. Concurrent disease is exceedingly frequent, and treatment aimed at such disease will afford symptomatic relief in many cases, thus confirming the wisdom of a conservative attitude.

As the majority of patients are overweight, reduction diets are important. At the same time the patient is advised to cease wearing tight corsets and to avoid exertion or reclining immediately after food. In addition a modified ulcer *régime* is prescribed, such as the frequent taking of small meals rather than large meals, mild sedation, and the exhibition of antacids and antispasmodics.

In simple stricture of the œsophagus, dilatation must be added to this routine. With this additional procedure the patient can frequently carry on in comparative comfort. In the treatment of elderly patients more active surgery should be avoided. In the treatment of the younger group resection of the stricture is probably preferable.

It would seem that the more drastic method of partial gastric resection to limit acid secretion is hardly indicated unless resistant duodenal or chronic gastric ulceration is an additional factor.

Obstruction, recurrent hæmorrhage or persistent disabling symptoms failing to respond to conservative treatment are indications for surgery, but in our experience are rare. Interruption of the left phrenic nerve has been disappointing.

Comment.

The symptomatology of thoracic stomach associated with hiatus herniation has been stressed, but it is necessary to emphasize that this lesion is as a rule an asymptomatic finding, and usually of no great importance. When symptoms do arise, the majority of patients will respond to conservative medical treatment. Above all, the correction of concurrent disease is of prime importance.

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LOBECTOMY FOR HYDATID DISEASE OF THE LUNG.

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Sydney.

LOBECTOMY has recently been advised as the primary operation of choice for hydatid cysts of the lung whether the cyst is simple or complicated. Most of the writers giving this advice live in countries where hydatid infection is rare; most surgeons from Australia, New Zealand and South America, where the disease is endemic, recommend conservative surgery because the results on the whole have been good.

Barrett (1947) has been the most persuasive advocate of resection, and he gives the following indications: (*a*) large cyst occupying most of the lobe, (*b*) cyst with tough adventitia, (*c*) cyst complicated by bronchiectasis or pulmonary suppuration, (*d*) dead cyst, (*e*) empty sac.

I have disagreed with some of these indications and have given my own indications (Susman, 1948 and 1950): (*a*) serious hæmorrhage from the cyst space during a conservative operation for simple removal of the cyst contents, (*b*) residual bronchiectasis, if symptoms warrant it, (*c*) empty sac, if hæmorrhage or infection warrants it, (*d*) when the diagnosis is uncertain, especially if there is a strong suspicion of carcinoma, (*e*) possibly a giant cyst, because of the risk of serious anaphylaxis if the cyst is attacked directly. In practice I have performed resection only for (*c*)—hæmorrhage or infection complicating an empty sac.

Since the publication of these papers, Waddle (1950) has reviewed 478 cases of pulmonary hydatid disease from the Hydatid Registry of the Royal Australasian College of Surgeons. (An editorial in THE MEDICAL JOURNAL OF AUSTRALIA, October 7, 1950, gives a good summary of all these articles.) From his study Waddle concludes as follows:

> There can be little justification for the operation of lobectomy in the primary treatment of hydatid cyst *per se* and the advocacy of such a drastic procedure does not accord with the requirements of any but an extremely small minority of cases, all of which have gross destruction of lung, which fact, and not the hydatid cyst, determines the need for removing the destroyed lobe.

The essential point of this statement is that it is change in the lung, not the cyst itself, that calls for resection. But, as will be seen from the pathological study of my three cases, the changes in the surrounding lung, though obvious enough, were not so gross as to warrant the term "destruction of the lung".

Waddle's review covers thirty years; the usual operation was simple removal of the cyst contents and drainage of the cyst space and/or the pleural cavity. Many of the operations were performed by occasional surgeons, and the total mortality of 6% must be regarded as satisfactorily low. Most of the survivors have been well and symptomless for an indefinite number of years; that is, the late morbidity is low, although Waddle states that bronchograms often show some residual bronchiectasis. Of the 478 cases reviewed by Waddle, in only four was resection carried out of part or whole of the lung, and in these the pathological changes were greater than in my three cases. What I want to emphasize is that, even with minimal damage to the surrounding lung, resection may be necessary because of infection or hæmorrhage.

It may be argued that primary lobectomy would have saved these patients from later troubles; but this argument loses force when it is recalled that the great majority of patients with hydatid cysts are cured by conservative surgery. Failure with conservative surgery almost always occurs when it is undertaken after the cyst has previously ruptured into a bronchus. The main lesson to be learnt is that conservative surgery should be carried out while the cyst is still intact, and I believe now that operation should be advised as soon as the diagnosis is made unless there is some obvious contraindication. Rupture of the cyst into the bronchus increases both the morbidity and the mortality rates.

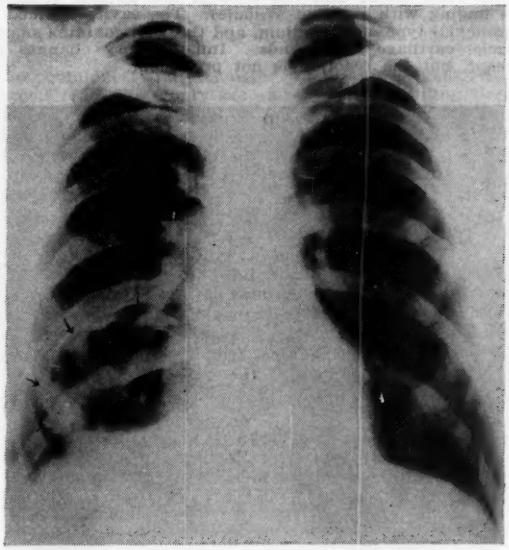

FIGURE I.
Case I: There is a cavity with fluid level at the base of the right lung.

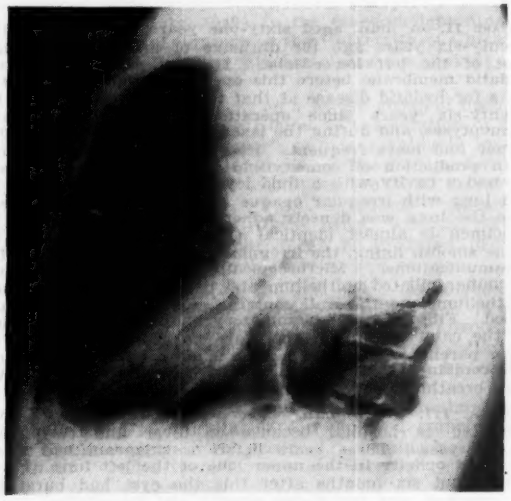

FIGURE II.
Case I: The lateral view shows the posterior position of the cavity.

Waddle states that "the tense pressure of the fluid contents of an unruptured cyst enclosed by the relatively thin friable ectocyst . . . renders its intact removal impossible". I would substitute the word difficult for impossible, because, in fact, it is possible to incise the adventitia without damaging the ectocyst and to remove the parasite intact; but it is doubtful whether the risk of rupture during this manœuvre makes it worth while. Most surgeons (and, I hope, physicians) must have recently been horrified to see advocated (under the misleading title of "Medical Treatment of Hydatid Cysts of Lung") a form of surgical treatment which should be unreservedly condemned (Castex and Capdehourat, 1950). It consists of repeated puncturing of the cyst, even if it is still uninfected, and the injection of penicillin and other substances, including formalin. Years ago the late Sir Louis Barnett, of

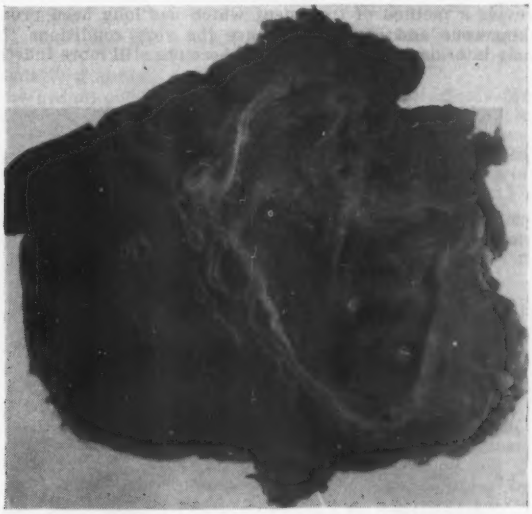

FIGURE III.
Case I: The lobe has been bisected to show the interior of the cavity.

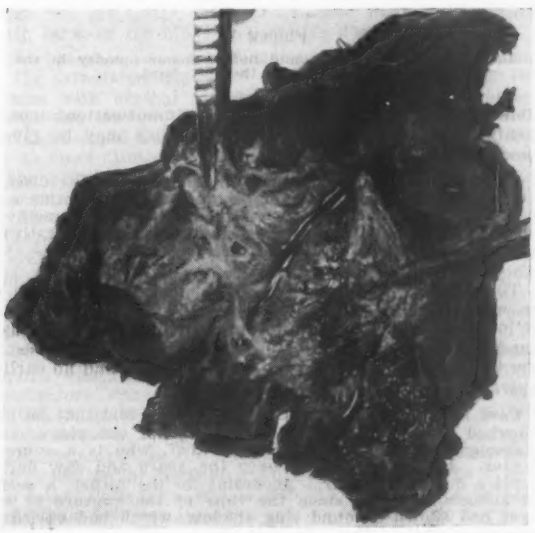

FIGURE IV.
Case II: The lobe has been bisected to show the interior of the cavity.

New Zealand, taught the dangers of putting formalin into pulmonary cysts, and Waddle has rightly questioned whether it is of any value under any circumstances (1950). The dangers of simple puncture, let alone of following this with injections, have been emphasized by many experienced surgeons of the past, and doctors must not be tempted to revive a method of treatment which has long been proven dangerous and can easily cause the very conditions that may later demand lobectomy. It becomes still more indefensible when we read that it is practised in out-patient departments and that as many as 30 injections may be given. Dew (1928) summed up thus:

> As a result of accumulated experience of the disease it became recognized that tapping was unscientific and unreliable and with the development of aseptic methods it has been totally abandoned in favour of comparatively riskless and efficient direct surgical attack on the cyst (page 226).

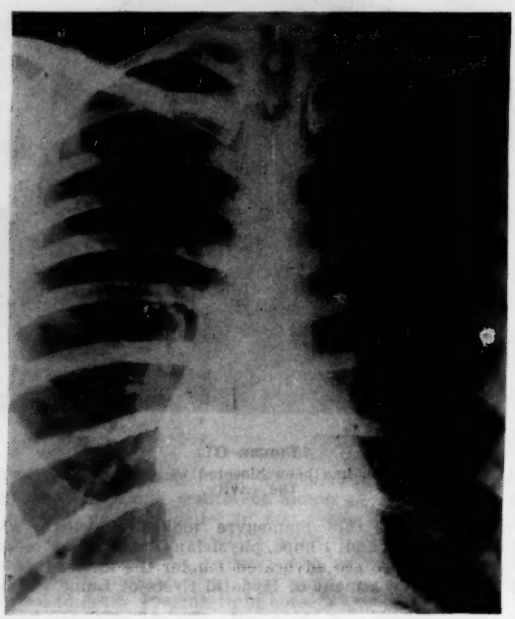

FIGURE V.

Case III: There is a round homogeneous opacity in the upper lobe of the left lung.

Three of my patients have needed lobectomy because of an empty sac complicated by infection and hæmorrhage. It is noteworthy that, in all three, the cyst had previously ruptured into a bronchus. One of them had had the resulting infected cyst drained; the other two had had no earlier operation.

CASE I.—A man, aged thirty-three years, said that he had coughed up a hydatid cyst at the age of ten years; this statement was confirmed by his father, who is a country doctor. He remained well over the years and saw active service during the war. According to the father, a series of skiagrams taken since the time of the rupture of the cyst had shown a round ring shadow, which had varied in size from film to film. In 1946 the patient had an hæmoptysis from which he quickly recovered. He remained well for the next three years, and then he had two hæmorrhages in a week. Since then he had been generally well, but had produced about an ounce of thick sputum daily. There were no abnormal physical signs, but skiagrams showed a well-defined cavity with a fluid level in the lower lobe of the right lung (Figures I and II). The Casoni test yielded a "doubtful positive" result. At operation the lung was found to be very adherent to the diaphragm and the chest wall. He made a good recovery after lobectomy, and he has remained well. Figure III shows the lobe after bisection. The cavity is seven centimetres in diameter, and it has a smooth wall thrown up into small folds; it was empty except for some small adherent blood clots. There is direct communication with the basal bronchi. The pleura over the cavity and the narrow strip of lung parenchyma between the cavity and the pleura are fibrotic. The rest of the lung tissue and the bronchi are normal (compare this finding with those of Waddle). The cavity is lined by a bronchial type of epithelium, and the wall contains smooth muscle, cartilage and glands. Inflammatory changes are present, but the reaction is not prominent.

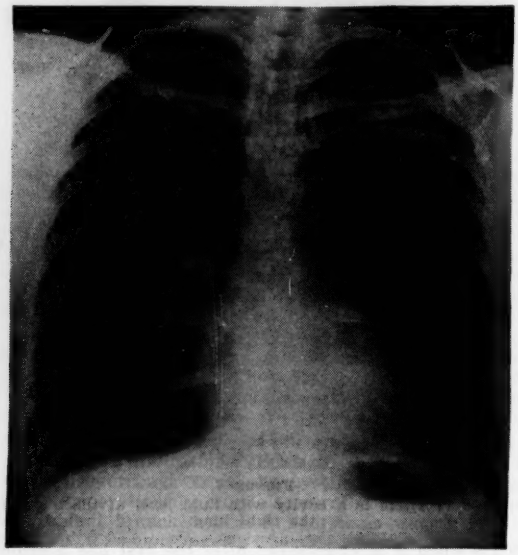

FIGURE VI.

Case III: The opacity is ill defined, without obvious skiagraphic evidence of cavitation.

CASE II.—A man, aged sixty-one years, had an operation twenty-six years ago for drainage of an infected hydatid cyst of the left lower lobe. He had coughed up some hydatid membrane before this operation, and the results of tests for hydatid disease at that time were positive. In the twenty-six years since operation he had had repeated hæmoptyses, and during the last two years they had become larger and more frequent. Recently he had had a cough with production of some viscid grey sputum. Skiagrams showed a cavity with a fluid level in the lower lobe of the left lung with irregular opaque areas around it. At operation the lung was densely adherent, as in Case I, and the specimen is almost identical (Figure IV); there are the same smooth lining, the irregular furrows and the bronchial communications. Microscopically the lining is partly columnar ciliated epithelium and partly stratified squamous epithelium, and the wall contains muscle with extravasated blood. Fibrosis and inflammatory changes are conspicuous in the cavity wall, but there are no gross changes in the lung parenchyma. The patient made a good recovery and has remained well; he volunteered the remark that he was less breathless than before operation.

CASE III.—A woman, aged twenty-three years, was admitted to hospital because of fever and two recent hæmoptyses. Three years before a skiagram had shown a round opacity in the upper lobe of the left lung (Figure V); about six months after this the cyst had burst into a bronchus, and the X-ray opacity lost its original roundness and definition. Two years later there was an irregular opacity with cavitation. The next skiagram, taken on the patient's admission to hospital with hæmoptysis, showed an ill-defined opacity without any obvious cavity (Figure VI). There were no abnormal physical signs, and the results of tests for hydatid disease were negative. The upper lobe of the left lung was removed, and the patient has been well in the five years since operation. The lobe contains an irregular cavity, five centimetres in its greatest diameter; it is half-filled with blood clot and debris; there are no recognizable hydatid elements. The wall is coated with

granulation tissue, and there is no epithelial lining. The surrounding lung is collapsed and fibrotic with thickened bronchi. (Compare Cases I and II, in which there was a well-developed epithelial layer and little change in the surrounding lung tissue.)

Summary.

The role of lobectomy for hydatid disease of the lung is discussed.

The commonest indication for lobectomy is hæmorrhage or sepsis from the empty sac and surrounding lung tissue, following rupture of the cyst into a bronchus.

Most hydatid cysts of the lung demand operation as soon as the diagnosis is made.

Conservative surgery will effect a cure in the great majority of cases.

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THE HEPARIN PROTAMINE TEST AND ITS CLINICAL SIGNIFICANCE.[1]

By NOEL M. GUTTERIDGE.

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THE heparin protamine test offers a new approach to the study of abnormal bleeding and provides a basis for effective therapy. The venue of this work was partly in Brisbane and partly (July, 1949) in the research laboratories of the Department of Surgery of the University of Chicago, where J. Garrot Allen and his associates are making an intensive study in this field.

This test throws a new light on a varied group of hæmorrhagic abnormalities. It contributes to the solution of the problem of the female "bleeder", and is a valuable investigation in thrombocytopenic purpura. The test, performed on the venous blood, indicates the presence of an excess of circulating anticoagulant. This substance resembles heparin, but is not necessarily identical with it. This abnormal condition of the blood can be produced in the dog and can be corrected both *in vitro* and *in vivo*.

The condition revealed by the test, with its correction by the intravenous administration of toluidine blue, is the basis of one of the three basic lines of treatment advocated by Park[(11)] for the aplastic anæmia of delayed radiation sickness following exposure to the atomic or hydrogen bomb.

The three basic forms of therapy are the administration of penicillin and of toluidine blue, and blood transfusion. Park's conclusions were the basis of a leading article in *The Lancet*[(10)] under the title "Atomic Defence".

Source of the Heparin.

According to Jorpes, heparin is produced by the granules in the mast cells in the connective tissue around the blood vessels.

Ehrich *et alii*[(7)] noted absence of thrombosis in a human scrotum weighing over 22 kilogrammes, surgically removed for elephantiasis. Numerous mast cells were seen in the neighbourhood of the blood vessels. From 10 kilograms of tumour tissue 11·1 grammes of crude heparin were extracted. On further purification, 126 milligrammes of pure heparin per kilogram of wet tissue were obtained. Ehrich *et alii* have given the name "heparinocyte" to the mast cell.

The mast cell has a small nucleus in relation to the size of the cell. The cell is outlined by closely packed metachromatic granules.

The granules in the mast cell have a special affinity for basic aniline dyes, of which toluidine blue is non-toxic in effective doses. It is this affinity which provides a means of neutralizing heparin by inhibiting the production of heparin. A more rapid method of neutralizing heparin is by the administration of protamine sulphate.

Hagedorn and Jensen's discovery that protamine stabilizes insulin, was extended by Chargaff *et alii*[(6)] to its effect on heparin. He found that protamine inactivates heparin immediately, both *in vitro* and *in vivo*.

Experimental Basis.

A workable conception of fluidity and coagulation of the blood is one of dynamic equilibrium between opposing factors. Some of them are paired. One pair is heparin and toluidine blue or protamine sulphate (Gutteridge[(9)]).

In the research laboratories of the Department of Surgery, the University of Chicago, and the Argonne National Laboratory, Chicago, as part of the Manhattan project, a study was made of the hæmorrhagic effects induced by exposure to ionizing radiation (Allen *et alii*[(4)]). In this study, dogs were exposed to 450*r*. Daily blood examinations up to a few days of death revealed, other than terminally, no loss or impairment of fibrinogen or prothrombin, vitamin *K*, calcium or phosphorus. Hæmorrhage externally and into every tissue of the body is the dominant feature of the irradiation syndrome. It begins about the fourth day, and the hæmorrhagic state frequently precedes, by several days, the inevitable fall in the number of platelets.

The red cell count, hæmoglobin value and total leucocyte count fell gradually, with progressive neutropenia, until death between the ninth and twelfth day after exposure.

The presence of an anticoagulant substance in the blood of the irradiated dogs was demonstrated by mixing the plasma with normal oxalated plasma, to which thromboplastin and calcium chloride had been added. It was shown that the delayed clotting times were due to active inhibition, not to mere dilution.

The possibility that this anticoagulant might have the nature of heparin was explored by mixing the plasma from irradiated dogs with substances known to inactivate heparin. Heparin, whose chemical composition is not definitely known, is possibly a mucoitin ester of polysulphuric acid. It may be bound by some basic dyes and proteins. Toluidine blue and protamine sulphate are both active in this respect. The addition of these substances returned the clotting time of the plasma of an irradiated dog to normal from its previous incoagulable state. From these in-vitro studies, a calculated amount of these heparin inactivators was then introduced intravenously into the animal, with the result that hæmorrhage ceased within twenty minutes.

The hæmorrhages throughout the body became absorbed, and at less lethal doses of irradiation the animal survived. The lethal dose for a dog is 300*r*. The dose used in this study was 450*r*.

Transfusions of citrated blood or injections of vitamin *K* or ascorbic acid did not materially affect the hæmorrhagic state of the lethally irradiated dogs.

The Principles of the Test.

A similar condition of varying degree can develop in the human. It is found that there are wide variations in the tolerance of human blood for added heparin. Once the amount of added heparin begins to increase the clotting time, very small increments then greatly prolong it. When

[1] Read at a meeting of the Section of Pathology, Bacteriology, Biochemistry and Experimental Medicine, Australasian Medical Congress (British Medical Association), Seventh Session, Brisbane, May-June, 1950.

these increments are plotted against the clotting times, the curve obtained for normal blood is a typical first-order curve (Allen *et alii*[3]). In order to make use of the more sensitive portions of the curve (Figure I), blood specimens are made incoagulable by the addition of a standard amount of heparin, and then "back titrated" with a standard heparin inactivator (protamine sulphate). The end point is remarkably constant with normal human blood. The figure in my series is 0·12 milligramme. The titration is influenced by an excess of this pathological heparin-like substance out of all proportion to the coagulation time.

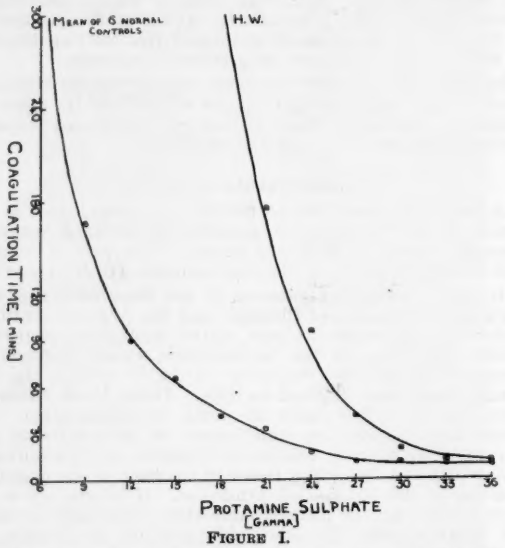

FIGURE I.

The curve resulting from the "back-titration" of specimens of blood rendered incoagulable with heparin. The addition of increasing quantities of protamine sulphate progressively neutralizes the heparin. The curve of the patient H.W. is also shown; his history chart is seen in Figure III.

The heparin-protamine test is therefore a measure of a fluidity coagulation factor similar to, though possibly not identical with, that produced by the presence of commercial beef heparin in the circulation. The result of this test may be increased by several factors, not all of which are known. It is necessary to study all the known factors concerned with fluidity-coagulation.

Clinical Significance.

While the nature of the clotting defect revealed by the test still lacks complete identification, a wide variety of hæmorrhage disorders have been shown to have a protamine titration higher than normal. This defect is usually corrected by administration of substances which inactivate heparin. It should be made clear at this stage that such inactivators are not considered to have any effect on vascular disorders. Dramatic improvement and control of external and interstitial hæmorrhage (such as petechiæ) have been frequently demonstrated, but this is regarded as a result of the control of the clotting defect alone. The defect is frequently associated with other abnormal conditions such as leuchæmia, the ultimate termination of which is not affected, but the associated hæmorrhagic state is controlled.

In those hæmorrhagic disorders in which the dominant abnormality is a raised protamine titration, good and sometimes permanent correction of the clotting defect usually takes place.

In more complicated hæmorrhagic conditions the response is limited to the extent to which the increased circulating anticoagulant affects the over-all clinical picture (Allen *et alii*[3]).

The individuals in whose plasma a raised protamine titration is found can be classified into the two following groups (Allen *et alii*[3]).

1. Patients without evident hæmocytologic disorders. This group is composed mainly of women suffering from menorrhagia, and some suffering from delayed post-partum hæmorrhage (Allen *et alii*[3]).

In the study of menorrhagia, the heparin-protamine test result appears to be a better index of a systematic disturbance than is either the coagulation time or the platelet count. The heparin-protamine test result was increased in 80 out of 106 determinations made during menorrhagic periods (Elghammer *et alii*[3]). A control series of women with normal menses showed only five increased titrations among 23 women studied. Moreover, the increases found were greater in the menorrhagic group than in the normal group. Pelvic examinations and biopsy studies were made in the menorrhagic group, and only two patients were found to have small uterine fibroid tumours. The diagnosis of menorrhagia was based on a history of prolonged profuse menstrual bleeding and anæmia.

A further control study by Elghammer *et alii* of 56 normal males revealed only three increased titrations among 102 determinations.

Previous to this study the only systemic clotting disturbances detected in menstruation had been a slight increase in the coagulation time, mild to moderate thrombocytopenia and some alterations in fibrinolytic activity. In the menorrhagic patients who were found to have an increased heparin-protamine titration, 25 out of 32 reverted to a normal menstrual period when given an intramuscular injection of 50 milligrammes of protamine sulphate on the second day of the menstrual period. The higher the titration the better the response to protamine titration. The effect was produced within four hours of the injection and continued during the remainder of the menstrual period. In the series of seven normal females studied in Brisbane, the result of the heparin-protamine test, carried out on the second day of the menstrual period, was normal in five and increased in two.

2. Patients with hæmocytological disorders. The patients in this group may be affected by (*a*) acute leuchæmia, (*b*) chronic leuchæmia, (*c*) post-irradiation syndrome, (*d*) Hodgkin's disease treated with nitrogen mustard therapy, (*e*) thrombocytopenia or (*f*) panhæmocytopenia.

The Technique of the Test.

This technique was developed by Allen *et alii*[3].

Reagents and Equipment.

The protamine sulphate stock solution is prepared as follows. Fifty milligrammes of powdered protamine sulphate are dissolved in distilled water and made up to 50 millilitres in a volumetric flask. Do not use the mixture for twenty-four hours. Keep it refrigerated at 1° to 4° C. (it is stable for one week).

Other essentials are as follows: liquid commercial heparin; a conical centrifuge tube, graduated to 15 millilitres; ten serology tubes (one centimetre inside diameter by eight centimetres long); 10-millilitre pipette, graduated to the tip; 20-millilitre syringe, glass-tipped, with a thin coating of light mineral oil; 18-gauge needles, one and a half inches long.

All glassware must be chemically clean and dry before being used.

Procedure.

The test is carried out as follows.

1. Pipetting the protamine solution. To each of the 10 serology tubes the protamine sulphate solution is added from a micropipette in increments of 0·02 millilitre (0·02 milligramme), beginning with 0·02 millilitre (0·02 milligramme) in the first tube and ending with 0·20 millilitre (0·20 milligramme) in the tenth tube. The entire 10 tube range runs serially from 0·02 millilitre to 0·20 millilitre (0·02 milligramme to 0·20 milligramme).

2. Pipetting the heparin into the conical tube. With a micropipette deliver 0·10 millilitre (1·0 milligramme) of liquid commercial heparin to the bottom of the conical centrifuge tube.

3. Venipuncture. Cleanse the skin with alcohol and dry. Apply a tourniquet and make a clean needle puncture. Gently aspirate (preferably by venous pressure) 12 to 14 millilitres of blood. If a good venipuncture is not accomplished on the initial puncture, another needle should be used, as tissue juice or small amounts of blood may introduce serious error. The syringe should be free of air bubbles.

4. Mixing the blood and heparin. Remove the needle from the syringe and allow the blood to run gently down the side of the conical tube containing 1·0 milligramme (0·1 millilitre) of heparin, bringing the blood to the 11 millilitre mark. Stopper the tube with a rubber stopper and slowly invert 15 times to obtain reasonable mixing. The remainder of the test should be completed within one hour. If the mixture is not pipetted at once, inversion mixing is repeated immediately before use.

5. Pipetting the blood. The 10 millilitre pipette is carefully filled with the heparinized blood mixture, aspiration of air bubbles being avoided. One millilitre of blood is allowed to flow down the side of each of the 10 tubes containing the protamine solution. Each tube is shaken briskly eight to ten times to obtain reasonable mixing of the heparinized blood with protamine solution. The entire series of tubes is then allowed to stand undisturbed for one hour at room temperature before the end point is read. The end point is defined in terms of the protamine content of the tube containing the least amount of protamine in which a solid clot has formed at this time.

Studies in Vivo.

It is rare not to have a response to therapy with toluidine blue when the response to the protamine titration test is raised.

There is no evidence that toluidine blue has any effect on hæmophilia or prothrombin deficiency due to dicoumarol therapy. Toluidine blue given intravenously in the therapeutic dosage of four milligrammes per kilogram of body weight diluted in 200 millilitres of isotonic saline has been administered by me on over one hundred occasions to patients of all ages and conditions, and the only side effects have been some transient nausea and occasional vomiting in about 10% of cases. It was reported to me in Boston, United States of America, that the administration in error of ten times the recommended dosage produced no ill effects. The dye is excreted in the urine and fæces for the ensuing four to seven days. It has been found that not every sample of toluidine blue is effective. I have been using a batch which was identical with a known effective batch in the University of Chicago and which I brought to Australia on my return.

Reports of Cases.

CASE I.—Mrs. I.A. was suffering from menorrhagia. She has always bruised easily, and this has been noted in her mother and maternal grandmother. Her son is also subject to bruising and spontaneous epistaxis. Since menstruation became reestablished after the birth of her only child two years and ten months ago, there has been an increasing blood loss, more pronounced for the past twelve months. On the first and second mornings of the menstrual period, no dressing would absorb the volume of blood lost. Two days before the penultimate menstrual period, an intravenous infusion of 200 milligrammes of toluidine blue dissolved in 200 millimetres of isotonic saline was administered. In the ensuing menstrual period a normal blood loss of four or five days' duration occurred. On the first day of the ultimate menstrual period, 50 milligrammes of protamine sulphate were given as an intramuscular injection. The remainder of the menstrual period was normal. Dr. H. McLelland made a pelvic examination of Mrs. I.A. Other than a small cervical scar, no evidence of abnormality was detected.

CASE II.—The second case is one of chronic lymphatic leuchæmia. Mrs. L.S. had noticed increasing weakness and pallor three and a half years earlier. A blood examination revealed lymphatic leuchæmia with a leucocyte count of 90,000 per cubic millimetre. Irradiation on two occasions has kept the leucocytes to about 20,000 per cubic millimetre. The associated anæmia has been controlled by blood transfusions every fourteen days over the past three years. During the last few months spontaneous bruising and bleeding from the gingivæ have been noticed. The heparin protamine test result was positive. Toluidine blue, 200 milligrammes, given with the blood transfusion controlled these symptoms.

CASE III.—The third case is one of thrombocytopenic purpura. R.A., a male patient, aged twelve years, weighing 38 kilograms, was referred on October 10, 1949, by the Brisbane Dental Hospital for investigation prior to the extraction of four teeth. He gave a history of frequent nose-bleeds and bruising for the preceding three years. In August, 1947, he had fallen astride the edge of the laundry tubs at his home, and had developed a large scrotal hæmatoma. He attended the out-patient department of the Brisbane Children's Hospital, where a blood examination gave the following results: hæmoglobin value 11·15 grammes per 100 millilitres, red blood cells 4,100,000 per cubic millimetre and leucocytes 8900 per cubic millimetre; no platelets were seen. In December, 1947, his platelets numbered 12,000 per cubic millilitre, and the Hess test result was recorded as "+++". In March, 1948, his platelet count was the same as in December, 1947, and his prothrombin index was 83% of normal.

In September, 1949, his platelet count was 3640 per cubic millimetre. His blood took fifteen and a half minutes to coagulate. The medical superintendent of the Dental Hospital, Mr. A. J. Hoole, to whom he had been referred for dental treatment, was not prepared to perform the necessary extraction of four teeth in the light of his hæmatological condition (see Figure II).

A protamine titration, estimated on October 10, was increased. On October 13, 148 milligrammes of toluidine blue in 200 millilitres of isotonic saline (four milligrammes per kilogram of body weight) were administered by the intravenous route. On the following day, the protamine titration had not completely returned to normal; accordingly a further intravenous infusion of 148 milligrammes of toluidine blue was given on October 17. The following day, his protamine titration was normal and his platelets had increased to 70,000 per cubic millimetre.

On October 19, at 11.30 a.m., his teeth were extracted at the Brisbane Dental Hospital under local anæsthesia. The medical superintendent reports as follows:

> The teeth extracted were
>
> $$\frac{ED \mid E}{6 \mid}$$
>
> The patient was treated as a normal patient, except that he remained for observation. At 12.30 p.m. all sockets with the exception of the mandibular right first molar had ceased bleeding. This socket showed signs of slight oozing of blood, which ceased at 3 p.m. with the aid of a sterile gauze pad. No further hæmorrhage occurred until after breakfast the following day, when the oozing recurred. This quickly responded to the patient's biting upon a sterile gauze pad for fifteen minutes. On this day petechiæ were visible on the soft palate; also one distinct lesion on the buccal mucosa retromolar triangular area. No further bleeding has occurred.

On February 16, 1950, his protamine titration was normal, and his platelets numbered 164,150 per cubic millimetre. On March 23, his platelet count had further increased to 224,000 per cubic millimetre. His mother reports that he has had only an occasional nose-bleed since his first treatment with toluidine blue. Bruising is also much reduced.

I am indebted to Dr. D. C. Fison, Medical Superintendent of the Brisbane Children's Hospital, for the details of this patient's record from August, 1947, to September, 1949.

CASE IV.—Case IV was one of panhæmocytopenia (aplastic anæmia). H.W., aged six years, had been a normal, healthy, active male child until three months before his admission to hospital. During that time the parents had noticed spontaneous bruising on his legs and body. He suffered from increasing generalized weakness, pain in the right upper quadrant of the abdomen, loss of weight and increasing pallor. For the previous few weeks he spent most of his time lying down. Five days previously, two of his lower temporary incisors had been shed naturally, and he had bled continuously from the shallow sockets. He was taken to the out-patient department of one children's hospital in Brisbane two days previously, and his mother had been assured that the bleeding would soon stop of its own accord. After two further days of bleeding, he was taken to another children's hospital, where he was admitted

on December 22, 1948. A hæmatological examination showed that his hæmoglobin value was 2·5 grammes *per centum*; the red blood corpuscles numbered 960,000 per cubic millimetre; the leucocytes numbered 600 per cubic millimetre; the platelets numbered 80,000 per cubic millimetre and the differential leucocyte count showed that 25% were neutrophile cells and 75% lymphocytes. The bleeding, coagulation and prothrombin times were normal. The report on a blood film was: "Resembles an aplastic anæmia rather than a leukæmia, no immature white or red cells seen." He was given a blood transfusion of 500 millilitres on the day of his admission to hospital. Thereafter, his hæmatological progress can be studied in the accompanying protocol (Figure I). A report on a marrow smear examination made on February 1, 1949, was as follows: "Supports a diagnosis of hypo or aplastic anæmia." X-ray studies on February 21, 1949, failed to reveal any bony lesions in the femur, tibia, humerus or skull. He received ten transfusions of blood during his four months' stay in hospital. During his stay he was confined to his cot, as any contact caused a bruise. He even bruised himself if he touched the side of his cot. The hospital authorities finally asked the parents to take the child home, as nothing more could be done for him.

H.W. came under my observation on April 26, 1949, when he was admitted to a private hospital in Brisbane. He had numerous large bruised areas on his body. Most of the incisions used for the ten previous transfusions showed surrounding bruising and some were oozing blood. The application to the upper part of his arm of a sphygmomanometer cuff, inflated to a pressure of 80 millimetres of mercury, produced a continuous pattern of petechiæ for a distance of about 12 centimetres in a distal direction along the arm. The petechiæ ceased abruptly at the distal edge of the sphygmomanometer cuff. Attempts at venepuncture produced an immediate hæmatoma. The heparin tolerance test showed the result given in Figure I.

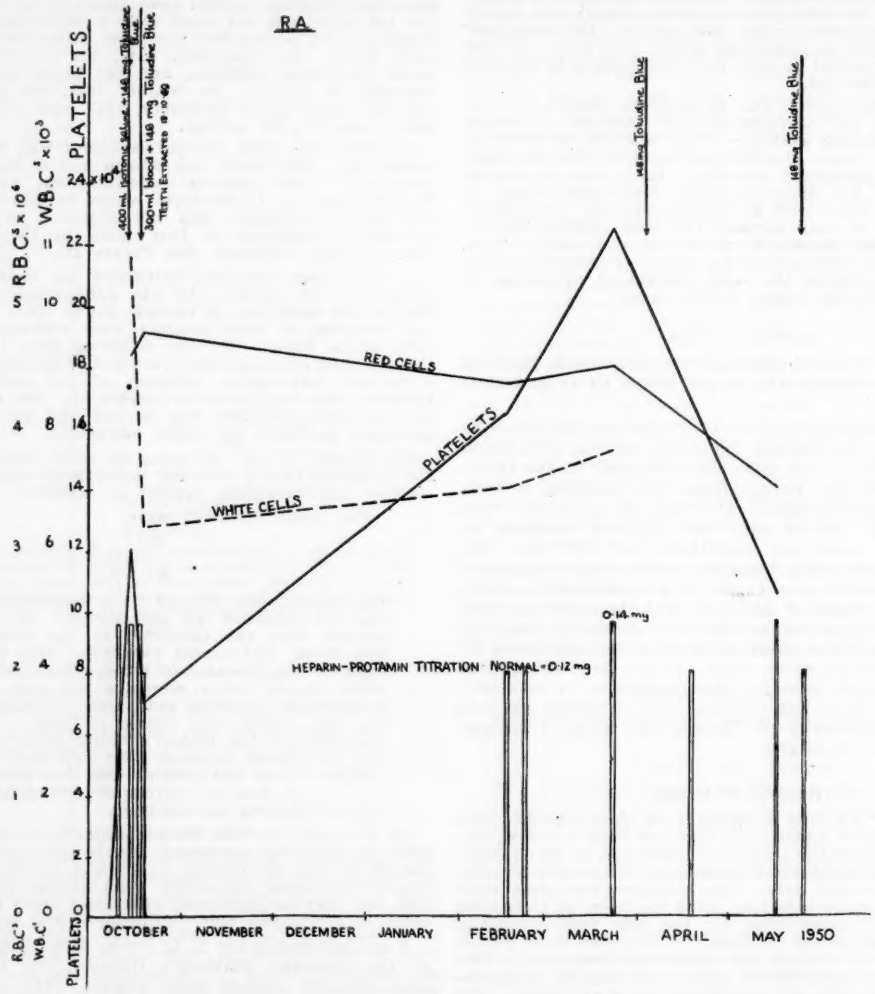

FIGURE II.

The history chart of R.A. (Case III—thrombocytopenic purpura). The response of his heparin-protamine titration to toluidine blue is demonstrated. His clinical condition was in parallel with his response.

On April 26, 1949, a blunt needle was introduced into a small vein on the plantar surface of his right foot, and four milligrammes per kilogram of body weight (84 milligrammes) of toluidine blue were administered, dissolved in 200 millilitres of isotonic saline. This injection was followed by a transfusion of 600 millilitres of blood. No untoward effects resulted from these infusions. The urine was coloured blue-green at the first passage after the infusion, and continued thus for the next six days. Within forty-eight hours the body bruises and petechiæ began to absorb, and three days after the infusion a second application of the sphygmomanometer cuff failed to elicit any petechiæ.

He was discharged to his home seven days after his admission to hospital and was able to live the normal active life of his age, without developing bruises. He returned to the private hospital on May 17 for two days for another blood transfusion and observation. He has not been in hospital since. He started school in June, 1949, and won his class footrace at the annual sports in December. As will be seen from his chart (Figure III), he has needed only four blood transfusions over the past seven months. On October 6, 1949, he had six teeth extracted under general anæsthesia. No abnormal bleeding followed the extractions. From April 21 to May 12 he suffered an attack of morbilli, from which he recovered without developing hæmorrhagic abnormalities, in spite of severe bronchitis associated with the disease. The interesting feature of his recovery is the return of his platelets to an almost normal level. His red blood cells have remained steady at about 4,000,000 per cubic millimetre for the past seven months. Examination of sternal marrow made in March, 1950, showed normal hæmatopoiesis.

I am indebted to Dr. D. C. Jackson for the details of this patient's history from December 22, 1948, to April, 1949.

Summary.

1. A new approach is offered to the study of hæmorrhagic disorders and complications. These include thrombocytopenic purpura, leuchæmia, menorrhagia and delayed post-partum hæmorrhage. A condition common to many hæmorrhagic states is an excess in the circulating blood of a heparin-like material.

2. The correction of the condition revealed by the test is one of the three basic therapies in the aplastic anæmia of delayed radiation sickness. This study, therefore, is an essential component of defence against the atomic and hydrogen bombs.

3. The technique of the test is described.

4. Clinical experience with the neutralization of the circulating excess of the heparin-like substance is given.

5. The following conditions are included in the illustrative case reports: (*a*) Menorrhagia. Excessive loss reduced to normal after premenstrual injection of toluidine blue. (*b*) Chronic lymphatic leuchæmia. Over three years the patient has received 70 blood transfusions. Bleeding from the gums and nose and subcutaneous hæmorrhages have been controlled by toluidine blue, following a positive response to the heparin-protamine test. (*c*) Thrombocytopenic purpura. The heparin-protamine test result was positive. Toluidine blue was adminis-

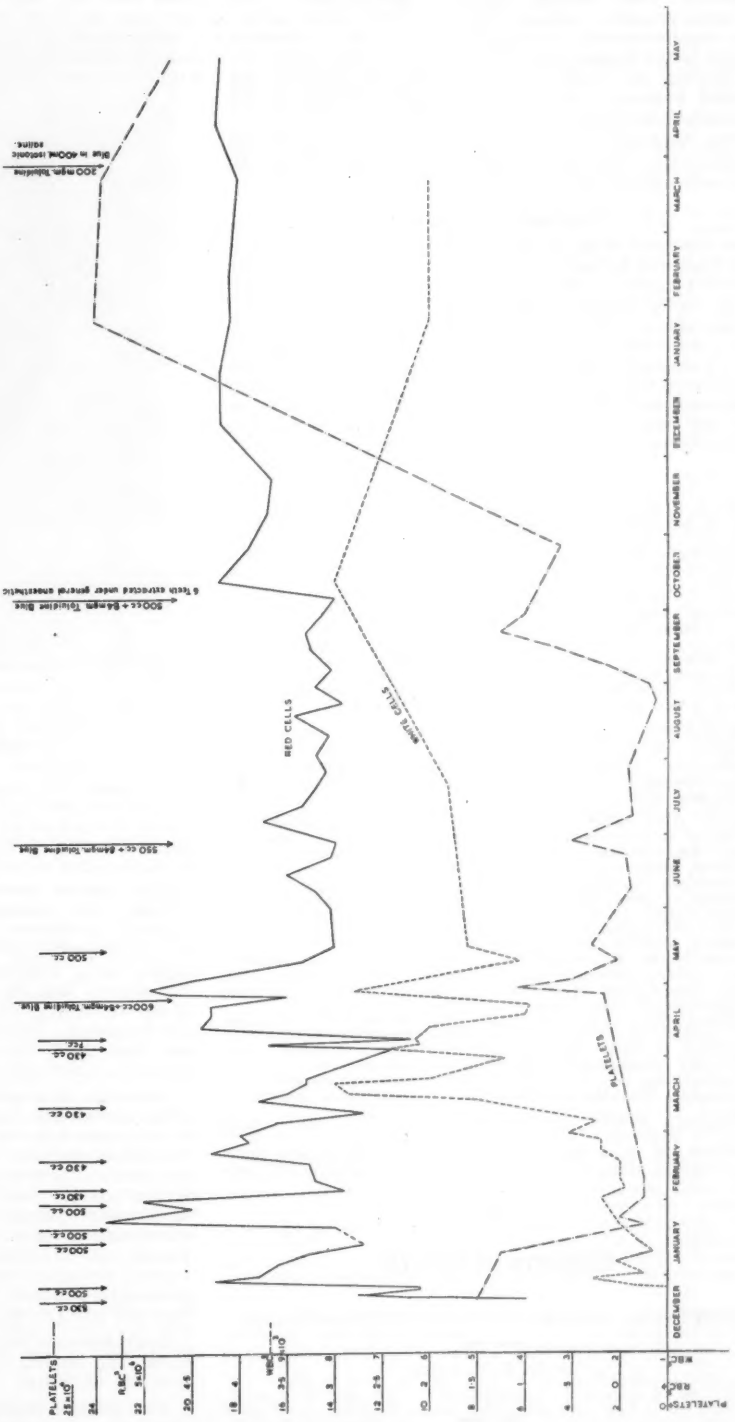

FIGURE III.
Chart of H.W. (Case IV—panhæmocytopenia, possibly aplastic anæmia).

tered. The platelets increased in number from 3640 per cubic millimetre in September, 1949, to 164,150 per cubic millimetre in February, 1950. Four teeth were extracted in October, 1949, without abnormal hæmorrhage. (*d*) Pan hæmocytopenia (aplastic anæmia) with gross hæmorrhagic manifestations. This patient's blood gave a positive response to the heparin-protamine test, and after toluidine-blue therapy all blood cellular components became and remained normal. No abnormal bleeding followed the extraction of six teeth.

6. The condition revealed by the test can be reproduced experimentally; but the exact cause and nature of the defect still awaits complete identification.

Acknowledgements.

I am indebted to Dr. J. Garrot Allen and Miss W. Egner, of the Research Laboratories of the Department of Surgery in the University of Chicago, for the technical training and clinical interpretation which was provided for me while I was working in their laboratories in July, 1949. The stimulating personality of Dr. Allen and his balanced judgement were an added benediction. Most of the technical studies in Australia were performed by Mr. F. G. Mills, biochemist on my staff, with the assistance of Miss F. M. Clarke. This study was made possible by the many practitioners who kindly referred their patients for investigation.

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Reports of Cases.

CARCINOMA OF THE BODY OF THE PANCREAS.

By C. W. R. PRICE,
Perth.

IN a recent article discussing the technique of excision of the body and tail of the pancreas, Cattell (1949) remarks that no carcinoma of the body suitable for the excision has as yet been seen at the Lahey Clinic. Sir Gordon Gordon-Taylor (1934), in reporting a seven-year cure in such a case, quoted extensively from the literature up to 1934 and noted that only four successful extirpations including his own case had been recorded up to that time. This argues a rarity sufficient to justify the reporting of the following case, in which there has been so far a three-year survival with very reasonable health.

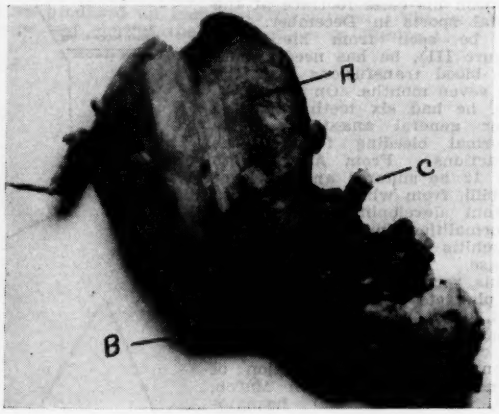

FIGURE I.

Photograph of specimen removed at operation. A, tumour; B, duct of Wirsung; C, splenic artery.

Clinical Record.

Mrs. E.B., aged sixty-four years, reported as an outpatient at the Royal Perth Hospital in January, 1948, complaining of upper abdominal pain of about five months' duration. She had suffered from flatulent indigestion for many years, especially after eating meat and eggs. The pain was described as being continuously present, worse after meals and radiating into the back. Anorexia was present and she had lost about fourteen pounds in weight during the preceding five months. Her bowels acted daily, she had noticed no abnormality in the stools and there was no disturbance of micturition. She had never had jaundice.

Examination showed the patient to be a pale, small woman; her hæmoglobin value was 11·4 grammes *per centum*. A tender, firm mass, rather less than the size of a table-tennis ball, was palpable in the epigastrium. It seemed to be slightly movable. An opaque meal examination showed that the mass lay medial to the lesser curvature of the stomach. Various other investigations did not clarify the diagnosis. Blood sugar and diastase estimations were not performed before operation. The provisional pre-operative diagnosis was "pancreatic cyst".

Operation was performed on February 26, 1948, under ether anæsthesia (Dr. R. V. Pratt). The mass was found to be situated in the body of the pancreas immediately to the left of the neck of the gland. After attempted aspiration had confirmed the solid nature of the mass, removal of the tail and body of the pancreas with the spleen was undertaken. Sharp dissection was necessary in a plane alarmingly close to the portal vein posteriorly, and as the photograph indicates (Figure I), the margin of excised normal tissue fell far short of the ideal. The cut end of the pancreatic remnant was oversewn with cotton and a drain was left down to it.

Convalescence was uneventful, with very little leakage from the wound, and the patient was discharged from hospital on March 13.

The diastase index was 60 units on the day after operation, but thereafter remained within the normal range. The highest blood sugar recording (while intravenous alimentation was still in use) was 188 milligrammes per 100 millilitres. Glycosuria was never found.

The gross specimen (Figure I) shows dilatation of the duct of Wirsung distal to the tumour to the calibre of a lead pencil.

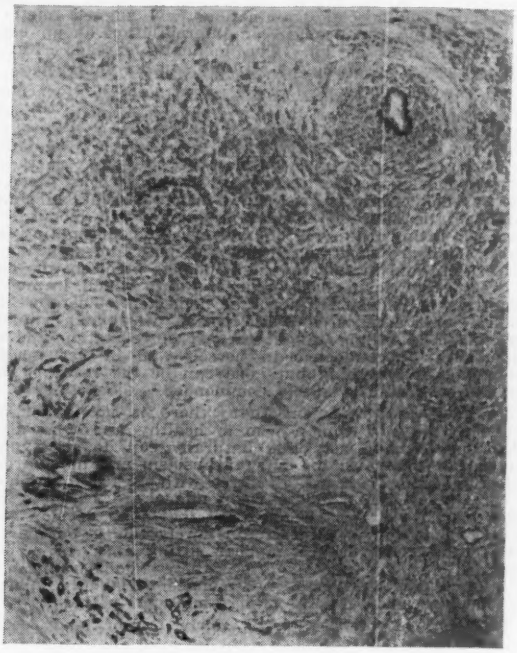

FIGURE II.

Photomicrograph of portion of the specimen, hæmatoxylin and eosin stain, × 100. (From the museum department, Royal Perth Hospital.)

Dr. T. R. Lubbe reported as follows on the microscopic features:

> Scirrhous carcinoma of the pancreas apparently originating from duct structures. There is much fibrosis and the intact islets are very conspicuous in the midst of fibrous tissue as well as in areas of tumour. The microphotograph [Figure II] gives a survey of the lesion. In the upper centre fibrosis is most marked, containing few remnants of tumour cells. In the periphery the irregularly outlined acini of the adenocarcinoma are seen. In the lower centre a small islet is present surrounded by tumour.

Mrs. E.B. has been examined as an out-patient at regular intervals since operation. By July 16, 1948, she had gained seven pounds in weight. She had noticed some intolerance of fatty foods and complained of lassitude. There was no complaint of diarrhœa or of offensive stools. In August, 1949, she complained of indigestion mostly occurring at night, and a barium meal X-ray examination was reported as revealing a "small prepyloric ulcer". After some alterations in her dietary and the occasional use of alkali, this indigestion has since remitted. When examined in February, 1951, she was well except for backache. X-ray examination of the lumbar part of the spine revealed some spondylitis only.

Acknowledgement.

This patient was treated in the wards of Mr. L. E. Le Souef, and I have pleasure in thanking him, not only for allowing me to operate upon her, but also for invaluable encouragement and advice during the course of the operation.

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Reviews.

MENTALLY AND PHYSICALLY HANDICAPPED CHILDREN.

As stated in the preface of "Children with Mental and Physical Handicaps", by J. E. Wallace Wallin, this book is the product of almost forty years of first-hand experience as director, supervisor and instructor in the fields of education and treatment of all kinds of handicapped children.[1] The work is a complete reference book for the treatment, whether remedial or educational, of every type of physical or mental disability.

On the mental side the various maladies, such as epilepsy, mongolism and pituitary infantilism, are all completely covered, and they are discussed from ætiological, psychological and clinical angles, and the possibility of educating children so afflicted is also gone into fully. The various theories of mental defectiveness are dealt with in some detail, and an important chapter is devoted to the prevalence of psychosis among intellectually normal and subnormal children.

All types of orthopædic handicaps are also included, and as some 42 pages are devoted to cerebral palsy, it can readily be seen that this condition is discussed at length. Poliomyelitis is looked at from all angles and the author appears to be favourably impressed with the Kenny technique.

At the end of each chapter a list of bibliographic references is given which will prove time-saving to serious readers, and the many case histories quoted throughout the work provide practical illustrations of the results achieved in treating the individual problems which unfortunately abound in handicapped children.

Psychiatrists, physiotherapists, educationalists and social workers will all find this work a useful addition to their bookshelves.

PNEUMONOKONIOSIS: RECENT DISCOVERIES.

THE proceedings of the Sixth Saranac Symposium which was held at Saranac Lake, New York, in 1947, have been edited by Arthur J. Vorwald and published in a handsome volume[2] entitled "Pneumoconiosis: Beryllium, Bauxite Fumes, Compensation". Nearly two hundred persons participated in the symposium, the deliberations of which underline the fact that with the introduction of new materials or processes into industry, or with changes in existing routine or methods, new health hazards are likely to arise. Further, the inclusion of pulmonary diseases of beryllium workers and of those exposed to fumes from bauxite under the heading of pneumonokoniosis illustrates the broader present-day scope of that term.

Although the discussions were limited mainly to three subjects, two of which concerned new and rather obscure diseases, it is a pity that unexpected difficulties prevented publication of the record until three years after the conference was held. In that period a number of investigations, no doubt instigated by the sessions at Saranac Lake, have been undertaken and their findings published. Thus, while this volume is fully authoritative as at the time of the meetings, it is now somewhat out of date, although the bibliography contains references to articles on beryllium which have appeared up to the beginning of the year 1950.

At the symposium nearly thirty papers on the "beryllium problem" were presented, and this subject occupies rather more than three-quarters of the published proceedings, under five main headings: the history and industrial aspects, the acute disease ("acute pneumonitis of beryllium workers"), the chronic or delayed disease ("pulmonary granulomatosis of beryllium workers"), toxicity studies in connexion with atomic energy production, and experimental aspects.

The contributions to this symposium (and subsequent studies) represent a concerted effort to solve in as short a time as possible, the many conflicting and complex prob-

[1] "Children with Mental and Physical Handicaps", by J. E. Wallace Wallin, Ph.D.; 1950. London: Staples Press, Limited. New York: Staples Press, Incorporated. 8½" × 5½", pp. 574, with illustrations. Price: 42s. net.

[2] "Pneumoconiosis: Beryllium, Bauxite Fumes, Compensation", edited by A. J. Vorwald, M.D; 1950. New York: Paul B. Hoeber, Incorporated. 9¼" × 6", pp, 676, with many illustrations.

lems involved in the discovery of a puzzling disease. It was therefore a triumph for industrial health when in June, 1949, major manufacturers of fluorescent lamps in the United States of America agreed to discontinue the use of beryllium phosphors, for it was in the fluorescent lamp industry that most of the cases of chronic lung disease had occurred.

Shaver's disease (called after Dr. C. G. Shaver), has not received the same degree of publicity as have the beryllium hazards, and it is accorded only forty-five pages in the conference report, chiefly in regard to the clinical and chemical aspects. This disease, which is none the less of serious import, occurs as a result of exposure to fume given off when a mixture of bauxite, iron and coke is treated in special electric furnaces for the manufacture of the abrasive corundum.

Compensation for occupational diseases, with emphasis on those in which the pulmonary tissues are involved, was discussed from the point of view of several interested parties, although trades unions were not represented. Some of the topics under consideration were: the extent of compensation coverage for industrial diseases, administration of compensation laws, employment problems in cases of non-disabling occupational disease, compensation for partial disability, preplacement and periodical medical examinations, the need for clear and definite statements in legislation, production incentives, sickness and welfare payment schemes, and the functions of the medical witness.

Because of its limited and rather specialized scope, most of this publication will be of particular interest to industrial physicians and hygienists, rather than to the medical profession as a whole. So far as is known, pulmonary disease attributable to beryllium or to bauxite has not occurred in Australia, although it is believed that beryllium phosphors are still being used in the fluorescent lamp industry here. Also it is possible that future industrial expansion in this country may involve risk of exposure to fume from bauxite, and so it is well for us to be aware beforehand of possible dangers. However, the section on compensation should have a more general and wider appeal. As an example of a first-class technical production, this volume could hardly be bettered.

FAINTING.

ENGEL in "Fainting, Physiological and Psychological Considerations"[1] has followed the discipline of the late Soma Weiss to whom this monograph, one of the American Lecture Series, is dedicated. The result is an embodiment of clinical experience and personal research with an emphatic regard of the psychological *milieu* of the symptom —fainting. As such the work, practical in intent, is addressed to the active physician as "a scientific basis for the understanding of syncope".

Fainting, a generic term, is defined as the resultant of three, possibly coexisting, basic mechanisms. The textual division of the subject is clinical—seven groups decided by common, multiple or obscure mechanisms. The groups are those due to a fall in arterial blood pressure, cardiac standstill, cerebral vascular disorders, cerebral metabolic disorders, hysteria, hyperventilation and heart disease. The first and largest group, including the commonest form—vasodepressor syncope—receives most attention and the following groups are in balance. Each sub-grouping is approached by definition; ætiology or the listing of proximate causes and therapeutic directions follow. However, treatment of hysterical and hyperventilation syncope is not contemplated. Vasodepressor syncope is viewed teleologically as a primitive response to danger; hysterical syncope is regarded as a conversion symptom with a sexual meaning—a content also of hyperventilation. Developmental confusions—that of chronic anxiety and its associated chronic hypotension with effort syndrome, that of hysterical syncope with the cerebral type of carotid sinus syncope, the lightheadedness in anxiety with hyperventilation—are indicated. After the systematic presentation are chapters on fainting during air travel—a correlation study—sudden death, incidence and diagnosis of syncope and, curiously, differential diagnosis. That fainting is but a symptom is insisted upon throughout. The references, seventy-eight in all, constitute an adequate introduction for fuller reading.

The monograph can well serve its intent. It will probably best stimulate the student wishing to evaluate the problem and here the limitation of the lecture series is inherent. That some may not agree with the stated psychogenesis of hysterical syncope is evident; that others would have a discussion of developed presyncope returning to full consciousness, and consequent anxiety, is probable. To many the section on special examinations will suggest methods of approaching the problem of differentiation as an essay in clinical investigation. The monograph read critically will further a physiological attitude toward diagnosis.

[1] "Fainting: Physiological and Psychological Considerations", by George L. Engel, M.D.; 1950. Illinois: Charles C. Thomas. Oxford: Blackwell Scientific Publications, Limited. 8½" × 5½", pp. 152. Price: 20s.

PREGNANCY AND LABOUR.

"INTRODUCTION TO MOTHERHOOD", by Grantly Dick Read, is a little book of ninety-two pages which presents in simple language the story of normal pregnancy and labour.[1] It aims at lifting the veil of mystery and dissipating the fears which are so often the basis of pain and suffering in parturition, and in this respect it defines precisely the duty of every obstetrician to his patient, a duty which, in modern obstetrics, should surely never be neglected. Therefore the woman who studies it will receive wise and valuable counsel. She will be taught the art of relaxation and the importance of confidence in herself and in her medical attendant, and nothing but good can be the result. Thus far the book can be highly commended.

However, the statement that "there is no longer any doubt that every woman who is healthy and who works and tries to have her child naturally can do so" typifies the author's extravagant superenthusiasm and must arouse objection. We shall not all agree with regard to euphoria in pregnancy that "unless their minds are balanced and they are happy to have conceived they may develop some nausea or morning sickness", for surely this is but part of the truth. Nor does it appear fitting that, in a book designed for the mother-to-be, there should appear in relation to the first emotional menace of labour: "This is the ideal occasion for 100 milligrammes of pethidin."

The care of the breasts is fairly taught, and feeding "on demand" advised.

In summary, one would commend this book to the woman in pregnancy, but would strengthen her faith by the promise that pain will be relieved when the need arises.

METHODS IN MEDICAL RESEARCH.

VOLUME I of "Methods in Medical Research" was a valuable contribution to medical literature by a number of well-known Americans and credit is due to the Governing Board—Irvine H. Page, A. C. Ivy, Colin H. MacLeod, Carl F. Schmidt, Eugene A. Stead and David L. Thomson. It contained sections on "Assay of Antibiotics", "Circulation—Blood Flow Measurement", "Selected Methods in Gastroenterological Research" and "Cellular Respiration".

Volume II of this series is now a welcome addition to our reference library and is commended to all who are interested in medical research.[2] It will help the laboratory worker most of all. This volume contains sections on "Methods of Study of Bacterial Viruses", "Pulmonary Function Tests" and "Assay of Hormone Secretions". The authors all enjoy world-wide renown in their particular fields. The section on bacteriophages will be of considerable interest, not only to the bacteriologist, but also to the geneticist and the clinician. Here it is pleasing to see that tribute is paid to the pioneer work of F. M. Burnet in Australia. The bacteriophage has proved to be a most valuable subject for study. In his introduction to this chapter, Mark H. Adams tells us: "Because of the ease with which they and their host may be grown and studied, because of the high precision with which they may be assayed and because a single infected host cell may be readily isolated and observed in the absence of interaction with other cells, the bacterial viruses have become the subjects of widespread research as models of the host-virus relationship. The bacterial viruses have been used as tools in the study of the synthesis of proteins and nucleic acids in infected cells, as a test system in the search for possible

[1] "Introduction to Motherhood", by Grantly Dick Read, M.A., M.D. (Camb.); 1950. London: William Heinemann (Medical Books), Limited. 8½" × 5½", pp. 104, with many illustrations. Price: 6s.

[2] "Methods in Medical Research", Editor-in-Chief, Julius H. Comroe, junior; Volume II; 1950. Chicago: The Year Book Publishers, Incorporated. 8½" × 5½", pp. 377, with a few illustrations. Price $6.50.

chemotherapeutic and antibiotic agents for virus diseases, in the study of the phenomenon of virus interference, in the study of the action of ultraviolet light and X-radiation on viruses and in the study of the mutational patterns of viruses and bacteria. The possibilities inherent in the bacteriophage as a tool in the study of the relationship between virus and host cell have been largely underrated. It is because of the greatly increased interest in recent years in this group of viruses that it was thought worth while to collect in one place the various techniques found most suitable for research in this field."

In the subsequent chapters detailed descriptions are given of the tests for pulmonary function and the assay of human secretions. The book is lucid and well illustrated throughout. Our American colleagues are to be congratulated on the excellence of their endeavours and we look forward to receiving Volumes III and IV, which are in preparation.

MEDICAL JURISPRUDENCE AND TOXICOLOGY.

"MEDICAL Jurisprudence and Toxicology", by John Glaister, Regius Professor of Forensic Medicine in the University of Glasgow, has reached its ninth edition.[1] This work was first published in 1902. The previous edition has been fully revised and brought up to date and new material has been added. The inclusion of new subject matter has been made necessary by changes in the law and progress in the field of forensic medicine. The law as set out is that of England and Scotland—it is not always applicable in this country.

The medico-legal aspects of artificial insemination are discussed. A short chapter has been included on the effects of the atom bomb, and there is additional information on the Rhesus factor. More substances, such as DDT, "Benzedrine" sulphate and radio-active materials have been included in the section on toxicology.

The text is well illustrated by photographs and of the 234 illustrations in this edition, 88 are in colour. Black and white illustrations of conditions observed on dead bodies sometimes do not convey a great deal, except to the eye of the experienced observer, but good colour renderings possess decided advantages.

Statements in text-books of forensic medicine about such subjects as *rigor mortis*, hypostasis, cooling of the body *et cetera* should be put forward as a general guide for the reader and interpreted as such. The time of onset and completeness of *rigor mortis*, for example, is different in nearly all the standard text-books. It is pleasing, therefore, to see in this book the avoidance of a positive statement and the use of sentences such as: "The period of invasion can only be stated in broad terms, there are so many modifying factors."

In the discussion on proximate causes of death a general statement is made on asphyxial deaths. When the author in a later chapter discusses the various asphyxial deaths such as drowning, hanging *et cetera* he refers the reader back to the general discussion on asphyxia. Instead of relying on this general statement about the time factor in asphyxia, it would be better to introduce a separate statement, for example, in the section on manual strangulation, as a further guide to the reader on the time taken to bring about death in this condition.

In the discussion on drowning, it is said that the presence of a fine foam or froth on the face may be regarded as presumptive evidence of drowning. This phraseology is good and more satisfactory than saying that the presence of foam or froth at the mouth or nostrils is characteristic of drowning, an expression sometimes interpreted by legal men as pathognomonic of drowning.

The blood chloride estimation in drowning is rather a disappointment as it so often fails to help the pathologist in the very case in which he needs extra aid in arriving at the cause of death, in those cases of drowning in which death has taken place some time previously. Workers in various parts of the world have found that there may be a difference in the chloride level of blood taken from the right and left sides of the heart when death was not due to drowning, and a difference in the chloride level of up to 50 milligrammes *per centum* in blood from the two sides of the heart does not necessarily indicate death from drowning.

It is interesting to note that very little is said about accidental suffocation of babies by bedding. Far too many cases of unexpected death from natural causes in infancy have been wrongly attributed to suffocation. The findings said to indicate an asphyxial death at autopsy are often seen in infants when death is due to natural causes.

At the end of each chapter there is a useful list of references to the subject matter discussed. There is a wealth of illustrative cases in the appropriate sections which are of much practical value to the reader. Some typographical errors are noted, but they do not detract from the general excellence of this publication. The work is monumental and the book has taken its place as one of the standard medico-legal text-books.

[1] "Medical Jurisprudence and Toxicology", by John Glaister, J.P., D.Sc., M.D., F.R.S.E.; Ninth Edition; 1950. Edinburgh: E. and S. Livingstone, Limited. 8½" × 5½", pp. 768, with many illustrations, some of them coloured. Price: 35s.

PRINCIPLES OF OPHTHALMOLOGY.

IT is nearly fifty years since Thomson Henderson published his first papers on the comparative anatomy of the filtration angle. "Principles of Ophthalmology" is a summary of his conclusions after a lifetime of research into the problems connected with the anterior segment of the globe.[1] An ardent Darwinian, he is convinced of the steady onward march of the organ of vision in its evolutionary progress, and gives no explanation for the strange lapse of the eye of the bat, tarsius, and the slow-moving loris.

A description of the comparative anatomy of the filtration angle is accompanied by a series of excellent original photomicrographs and drawings which clarify many of the difficulties associated with the study of this region. In view of Henderson's painstaking work on the pectinate ligament, it is to be regretted that it was to be left to another observer to suggest that the iris process in the human eye is the remnant of this structure. He gives very clear reasons for his belief that the intraocular and intracranial pressures are the same. This equilibrium is maintained by the free passage of aqueous from the anterior chamber through the spaces of the cribriform ligament to the suprachorioidal space of the eye, and thence backwards through the interstices of the *lamina cribrosa* to the subdural space of the brain. The possibility of the passage of aqueous from the anterior chamber to the suprachorioidal space is not supported by the behaviour of neoplasms in this region. Infiltrating tumours of the iris spread through the spaces of the cribriform ligament and along the perivascular spaces of the penetrating vessels, but show no tendency to spread backwards into the suprachorioidal space. Newgrowths occupying that space do not tend to infiltrate forwards into the spaces of the pectinate ligament. Nevertheless, the author maintains that to assert that the intraocular pressure is higher than the intracranial is to violate all physiological principles.

His interpretation of the mechanism of accommodation is supported by anatomical observations which he uses to demolish the theories of his opponents whom he denounces as the perpetuators of error, the modern counterparts of the mediæval copyists.

The book concludes with a study of the pathology of the cataract incision, and of his methods for the treatment of hypopyon ulcer, glaucoma and cataract. Henderson's heroic use of the keratome instead of the Graefe knife puts to shame the up-to-date surgeon who nibbles away at the limbus with scissors. This is a volume for the critical ophthalmologist.

[1] "Principles of Ophthalmology", by Thomson Henderson, M.D.; 1950. London: William Heinemann (Medical Books), Limited. 8½" × 5½", pp. 236, with 63 illustrations. Price: 20s.

BLOOD GROUPS IN MAN.

SINCE the first announcement of the impending publication of "Blood Groups in Man" by R. R. Race and Ruth Sanger,[2] those interested in this subject have impatiently awaited its appearance. If they have not yet acquired copies, they can be assured that they will not be disappointed—rather are they likely to be completely satisfied or perhaps even dazzled by the quality of this small book.

The enormously intensified interest during the last ten years in the blood groups, initially set in train by the discovery of the Rh factor, has led to such an outpouring of "literature" that even some of those whose work is in this sphere despair of being able to keep up with and evaluate

[2] "Blood Groups in Man", by R. R. Race, Ph.D. (Cantab.), M.R.C.S. (England), and Ruth Sanger, Ph.D. (London), B.Sc. (Sydney), with a foreword by Professor R. A. Fisher, F.R.S.; 1950. Oxford: Blackwell Scientific Publications, Limited. Price: 30s.

it. But within the compass of only 290 pages, and withal in pleasantly large type and with well-spaced arrangement, Race and Sanger have brilliantly given within their self-imposed limits an amazingly clear and eclectic account of current knowledge on this complex subject. Where detail is helpful, as in descriptions of relevant technique especially in connexion with the Rh system, and in tabulated information, it is in this book, but by the skilful method of its inclusion, it does not obscure the main theme, which, as the authors note in their preface, is concerned with the blood groups and their inheritance.

One or more chapters are devoted to each of the eight well-established blood group systems, ABO, MNS, P, Lutheran, Kell, the secretor and non-secretor status in the ABO system, Lewis and Duffy, and other very rare groups are briefly mentioned. There are also short but useful chapters on blood groups and disease, and on blood groups and problems of parentage. But in keeping with the authors' purpose, the greater emphasis is on those aspects of our knowledge of the blood groups which are of most importance in genetics, as, for example, interpretation of the data provided by serological investigations in relation to the method of inheritance of the various factors, and for the computation of gene frequencies. This work, for much of which Race and Sanger are themselves responsible, and which they expound with such masterly simplicity, opens up wide horizons. There seems no doubt of the vast possibilities of blood grouping as a tool, probably the principal tool of the future, in the study of human genetics.

RECENT ADVANCES IN OCULAR PROSTHESIS.

"RECENT ADVANCES IN OCULAR PROSTHESIS", by Mr. H. Prince, is a companion volume to his book "Ocular Prosthesis" and provides a great deal more information on this subject which is of practical assistance to the ophthalmic surgeon.[1] The author has consulted various surgeons who are interesting themselves in the field of ocular prosthesis and consequently is able to describe the surgical techniques involved in the fitting of various types of implant. The book is abundantly illustrated and demonstrates clearly the various stages of operation.

Renewed interest in implants has arisen since the advent of materials regarded as physiologically inert, such as acrylic, tantalum and vitallium, and those who are anxious to acquaint themselves with recent work in ocular prosthesis would do well to consult Mr. Prince's new book on this subject.

"MAN IS A MICROCOSM."

DR. J. A. V. BUTLER, of the Chester Beatty Research Institute, Royal Cancer Hospital, Fulham, London, has written a book, "Man is a Microcosm", which can be warmly commended.[2] At a time when popular concepts in medical science are concerned with wonder drugs, test tube babies and electronic brains it is refreshing to find an exposition in graceful English of human physiology and biochemistry which is scholarly and which displays candour, restraint and sound judgement. Popular books often suffer from one of two faults—either the author airs views which are peculiarly his own or else he has distinguished himself in a very small field, but has been approached by a publisher to write a book dealing with the whole terrain of the science named. From both these faults this volume is happily free. After a careful presentation of newer knowledge concerning proteins, enzymes, hormones, trace elements, cellular activity, muscle, nerve and brain the book ends with a philosophical treatment of action and free will, speech and thought and "the stature of man". The main thesis of the volume is that whilst modern researches in the physical sciences are tending ever towards unification or at least towards simplification, the opposite is true of biology; here the latest investigations have opened up more and more complexities in life and have assured man that he is something better than an inhabitant of a second-rate planet revolving round a second-rate star; he is a veritable world or microcosm of wonder-arousing mechanisms and adjustments. Even experts will find something new and well worthy of study in this work.

[1] "Recent Advances in Ocular Prosthesis", by J. H. Prince, F.B.O.A., F.S.M.C., F.R.M.S., F.Z.S.; 1950. Edinburgh: E. and S. Livingstone, Limited. 8½" × 5½", pp. 164, with 89 illustrations. Price: 20s.

[2] "Man is a Microcosm", by J. A. V. Butler; 1950. London: Macmillan and Company, Limited. 7¾" × 5¼", pp. 168, with some illustrations. Price: 10s. 6d.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Introduction to Diophthalmology", by N. A. Stutterheim, M.D. (Rand.), with a foreword by W. P. C. Zeeman, M.D.; 1950. London: H. K. Lewis and Company, Limited. 8½" × 5½", pp. 56, with two graphs. Price: 7s. 6d.

The author proposes a new theory for immediate depth perception.

"Pioneer Doctor: A Biography of John Singleton", by Mary Kent Hughes; 1950. Melbourne: Geoffrey Cumberlege, Oxford University Press. 8½" × 5½", pp. 170, with some illustrations. Price: 10s. 6d.

John Singleton was the author's great grandfather.

"Muscular Contraction: A Topic in Molecular Physiology", by W. F. H. M. Mommaerts, Ph.D.; 1950. London: Interscience Publishers, Limited. New York: Interscience Publishers, Incorporated. 9" × 6", pp. 212, with a few illustrations. Price: $4.20.

Describes experimental discoveries in the biochemistry of muscle and their immediate interpretations.

"A Guide to Medicine", by Ivo Geikie-Cobb, M.D., with special articles by various contributors; 1950. London, Sydney, Toronto and Bombay: George G. Harrap and Company, Limited. 9" × 6", pp. 420. Price: 23s. 6d.

Is not a text-book or an encyclopædia, but a "guide" to those who wish to know the meaning of medical words.

"Regional Orthopedic Surgery", by Paul C. Colonna, M.D.; 1950. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 9¾" × 6¼", pp. 736, with 474 illustrations. Price: £5 9s. 3d.

An attempt has been made to deal with common clinical conditions and to stress the principles of physical diagnosis and treatment.

"Understanding Natural Childbirth: A Book for the Expectant Mother", by Herbert Thoms, M.D., in collaboration with Laurence G. Roth, M.D., with a picture story by David Linton; 1950. New York, London and Toronto: McGraw-Hill Book Company, Incorporated. 10" × 7", pp. 124, with many illustrations. Price: $3.50.

Deals with the natural childbirth "program" as it operates at the Grace-New Haven Community Hospital, United States of America.

"The Year Book of General Surgery (July, 1949-June, 1950)", edited by Evarts A. Graham, A.B., M.D., with a section on Anæsthesia, edited by Stuart C. Cullen, M.D.; 1950. Chicago: The Year Book Publishers, Incorporated. 7¾" × 5¼", pp. 692, with many illustrations. Price: $5.00.

One of the "Practical Medicine Series" of year books.

"The Medical Works of Hippocrates", by John Chadwick, M.A. (Cantab.), and W. N. Mann, M.D. (Lond.), F.R.C.P.; 1950. Oxford: Blackwell Scientific Publications, Limited. 8½" × 5½", pp. 316. Price: 20s.

"A new translation from the Greek made especially for English readers."

"Eternal Eve", by Harvey Graham; 1950. London: William Heinemann (Medical Books), Limited. 10" × 6", pp. 726, with many illustrations. Price: 42s.

The story "concerns a million women and a handful of men"—great men and outstanding women, with midwives and charlatans, man-midwives and quacks.

"Bronchoesophagology", by Chevalier Jackson, M.D., Sc.D., LL.D., F.A.C.S., and Chevalier L. Jackson, M.D., M.Sc., F.A.C.S.; 1950. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 10" × 7", pp. 380, with 260 illustrations. Price: £5 18s. 9d.

An attempt to present the current concepts on the subject and to retain tried methods of technique.

The Medical Journal of Australia

SATURDAY, MARCH 17, 1951.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

ANOTHER REPORT ON NURSING.

DURING the past few years reference has been made in these columns to several important publications on the recruitment and training of nurses. On September 25, 1943, a study by Sheila M. Bevington on nursing life and discipline was mentioned. This study was based on over 500 interviews. On November 29, 1947, discussion centred round the report of a working party on the recruitment and training of nurses; the report was published by the Ministry of Health and the Department of Health of Scotland. On January 8, 1949, a minority report of the same working party was discussed, and on April 2, 1949, mention was made of a report prepared for the National Nursing Council by Esther Lucille Brown, Ph.D. No apology need be made for repeated reference to the subject of nursing. As most readers are aware, this journal has for some time advocated the setting up of a special committee in Australia to inquire into the subject of nursing and the recruitment of nurses from the Australian point of view. The latest report comes from the World Health Organization.[1] The Expert Committee on Nursing consists of seven members and three coopted members. The chairman is Miss M. I. Lambie, who was formerly Director of the Division of Nursing, Department of Health, Wellington, New Zealand. The other members come from India, France, Chile, Washington, Finland, United Kingdom and Geneva. The terms of reference of the committee, which held its first meeting in February, 1950, were as follows: (i) to advise the World Health Assembly on measures to ensure the recruitment of nurses in proportion to the needs of each country; (ii) to advise the World Health Assembly on measures to give nurses training in keeping with the numerous and complicated tasks which will devolve upon them. In the introductory statement the reader learns that the lack of nursing personnel is world-wide. The number of nurses varies in different countries from none whatever for millions of people to those which have one nurse for approximately every 400 persons. The ratio of one to 400 applies in countries which have highly organized health services, but even here hospital beds are unused and new health programmes are impeded for want of nurses. Nurses are needed in greater numbers than any other categories of health workers, because they have direct individual and lasting contact with people who are ill and well. In this sense, it is stated, nurses are the final agents of the health services.

The committee decided that the provision of a nursing service of adequate quantity and quality required three simultaneous and related approaches. These were: (*a*) the securing of candidates for training of all types; (*b*) the promotion of the most effective use of various types of nursing personnel; (*c*) provision of educational facilities and programmes for all types of nurses needed. We do not propose to deal with the whole of this report, but rather to make reference to those portions which have bearing on Australian conditions. At the same time it is interesting to note that the stage of development of nursing is stated to vary greatly from culture to culture. Its development is limited by the stage of development of medicine and public health. In those countries where medicine is highly developed and nursing is not, the health status of the people does not reflect the advanced stage of medicine. Nursing is thus held to be essential to the "vitalization" of the health programme. This is probably correct; but it must not be taken as indicating a superiority of nursing over the other factors leading to advancement of health. The numbers and quality of medical practitioners, nurses, administrators and others connected with the health services are all equally important. If one is deficient, the whole becomes deficient. This is probably one of the most cogent reasons that can be advanced for the study of nursing in our own or any other country.

The education undertaken by a nurse during her training is known in this report as "basic nursing education". The committee names several points in the basic education of nurses which call for special emphasis in countries which have nursing programmes of recent origin, or in which nursing education is now being established or revised. In the first place the school of nursing should be directed by a nurse competent both as a nurse and as an educator. In the second place, while medical practitioners and other professional and health workers should give instructions to student nurses in nursing schools, instruction in nursing in clinical fields and supervision of practice in these fields should be in the hands of qualified nurses. In the third place the programme of every basic school of nursing should include the integration of the principles of mental health, public health and prevention of disease, in order to prepare the nurse for her future teaching of preventive medicine. In the fourth place candidates for admission to nursing schools should be carefully selected. Among the means recommended for selection are the use of standardized tests and interviews by psychological and psychiatric experts. Ideally this has much to commend it. In Australia, however, there seems to be a tendency to lower the educational standard of recruits to the nursing profession, no doubt because it is so hard to secure recruits. The preservation of health, in which members of the nursing profession have to take an important part, calls for psychological insight, wisdom and knowledge. These qualities are not likely to be attained if the trainee has an insufficient standard of

[1] Expert Committee on Nursing, Report of the First Session; World Health Organization, Technical Report Series Number 24. Geneva, November, 1950. 9" × 6¼", pp. 30. Price: 1s. 6d.

general education on which to base her special nursing attainments. The profession of nursing has many grades of activity, and here what the authors of the present report call the "post-basic education" of nursing is of importance. They hold that this post-basic education has three distinct objects: (i) to prepare for work in a field which at present is not included in basic nursing education; (ii) to keep nurses up to date in modern developments by means of refresher courses in the different fields of nursing; (iii) to provide advanced or specialized preparation for senior positions in hospitals, schools of nursing and public health. We may consider these in the reverse order. In the past there were no special courses of instruction for nurses who wished to proceed to the higher positions and to become matrons and so on. Nurses stayed on in the hospitals in which they had been trained and were advanced in the hospital's service if they seemed likely to be able to fill a required position with success. Such persons as tutor sisters were not known. Nowadays nurses can undertake special courses of training in administration and in the teaching of nursing. They can obtain certificates or some form of official recognition in both these spheres. Special types of nursing training, of course, are in existence. These include mental nursing, children's nursing, obstetric nursing and so on. The provision of refresher courses for nurses, however, leaves much to be desired. It should be possible for a nurse to take a refresher course in any particular aspect of nursing in which she has already been trained. Further, it would be desirable that she should do so from time to time. The preparation for work in a field which at present is not included in basic nursing education is a need which, in the opinion of this committee, should tend to disappear as programmes of basic nursing education attain a higher level. One aspect of nursing to which more attention should be paid is industrial nursing. Admittedly, a woman who has undergone a course of special training before registration as a nurse can, if she has a reasonable amount of common sense, give a good account of herself in any sphere of nursing which she cares to take up. Against this it must be admitted that success in any sphere is more likely to be attained if a person entering it has had some special training than if common sense only in addition to basic training is the sheet anchor. In regard to industrial nursing, the following statement is quoted. It was made by the Expert Committee on Professional and Technical Education:

> It was agreed that every general practitioner and every nurse should have some knowledge of industrial health. . . . The view was expressed that industrial health should be regarded as an essential part of public health and that isolated courses of training in the industrial branch for persons without public health training should be discouraged.

The authors of the present report agree that industrial nurses should have preparation in public health nursing. The committee makes special reference to training in anæsthesia and in other specialties such as laboratory work, physiotherapy and so on. These do not concern the nursing profession in Australia. One important recommendation made by the committee is that, in order to develop essential nursing leadership in countries, national governments should be urged to award an increasing number of fellowships to well-selected nurses for study in educational centres.

Reference to the report of this committee will be concluded with a mention of its recommendation that the World Health Organization should urge each member government to undertake a study of: (*a*) the existing supply of each type of nursing personnel and of various types of nursing personnel; (*b*) the estimated number of each type of nursing personnel needed in all categories of employment, based on existing and prospective health programmes; (*c*) the factors which interfere with securing candidates for training of various types; (*d*) the effectiveness with which nursing resources are used. Some day someone somewhere in Australia will move to have this done. If it is done, nothing but good can follow. The other points which arise from this report, and which should at least be discussed by those interested in nursing in this country, are the provision of refresher courses for nurses, the paying of increased attention to industrial nursing, and the making of some sort of move to provide fellowships for nurses in educational centres in different parts of the world.

Current Comment.

DRUG THERAPY IN DISSEMINATED SCLEROSIS.

THE treatment of disseminated (or multiple) sclerosis continues to be baffling. The literature on the subject is considerable, and beneficial effects have been claimed from various types of therapy. The value will probably not be questioned of some symptomatic treatment and of measures, particularly psychological, aimed at helping the patient to cope with the disease and maintain his morale. Much is being done along these lines and more can be done; in Australia the matter has probably not received the attention it merits. However, this is conditioned by the availability or otherwise of a specific means of cure or amelioration. A thorough review of drug therapy prepared for the Medical Advisory Board of the National Multiple Sclerosis Society in the United States by George A. Schumacher[1] indicates that the specific remedy has not yet come to light. There would be little point in retailing here Schumacher's review of the literature and his careful consideration of drugs that have been suggested or advocated. He divides them into two groups: those aimed at "the prevention (or improvement) of lesions" and those aimed at symptomatic relief. In the first group he places vasodilators, anticoagulants, circulatory stimulants, vitamins, drugs aimed at modifying allergic states, and miscellaneous agents such as cytochrome C, trypan red and vaccines. In the second group are drugs claimed to alleviate muscle spasticity (quinine, neostigmine, curare and mephenesin) and agents directed towards the relief of miscellaneous symptoms. Schumacher's conclusion, in summary, is that the outlook for cure of the disease by use of drugs is unpromising and that the outlook for symptomatic relief by drugs is less optimistic than would appear from the large number of reports which make claims of favourable effects. The claims for both types of drug therapy, he states, would seem in many instances to be unwarranted. Frequently the approach to drug therapy, with several notable exceptions, has not been scientifically sound. In general the studies are insufficiently documented, and both rationale and conclusions are based on insufficient precise scientific data. Conflicting and incompatible data with resultant opposing theories appear in the works of different and the same investigators. Schumacher's view is that all hypotheses of effective drug action are presumptive so far as they are not controlled by measurements of the

[1] *The Journal of the American Medical Association*, August 5, 1950.

primary pharmacodynamic action of the drug in the patient, or so far as they rest on theories of pathogenesis (of lesion or symptoms) which in themselves are presumptive. From his survey he concludes that no case of unequivocal cure of disseminated sclerosis is on record. No case of complete relief of symptoms of significant duration has been proved to be due to a therapeutic measure. No study statistically summarized advances a sufficiently high percentage of good results in a significantly large number of cases to warrant general use of the method in question by practising physicians—except as a means of further experimental investigation, and with only some of the agents that he mentions, is further experimental work considered to be indicated. Schumacher quotes from a personal communication the finding of A. R. Maclean that in a series of 416 patients with disseminated sclerosis followed for an average of fifteen years, 4% to 6% became disabled in terms of either walking or working ability during the course of any one year; he suggests from this that one of the criteria to establish proof of the efficacy of any form of treatment in disseminated sclerosis is an incidence rate of disability (in terms of walking or working) no greater than four in a group of 100 patients followed for one year. This seems logical and in conformity with the need for exacting standards in assessing drugs for the treatment of this disease. Schumacher's review has the dampening effect of a shower of rain, but in the same metaphor it clears the air; only care will prevent the reaccumulation of unwarranted claims and the raising of false hopes. There seems to be no warrant for complete despair of specific drug therapy for disseminated sclerosis, but for the present the most helpful treatment appears to be on the lines of aiding those affected to prolong their period of useful active life. This, as was recently pointed out by Jean Macnamara,[1] is a field of usefulness waiting for physiotherapists.

THE RABBIT PROBLEM.

PRACTICALLY everyone in Australia has heard of the rabbit problem, though probably few outside the areas affected have any idea of what it means in practice—of the damage done, of the money and labour expended in control measures, and of the potential devastation kept under control only by such measures. In actual fact a good deal of success has been achieved in the past in rabbit control, but the unavoidable neglect of the problem in wartime with post-war difficulties, especially in such matters as availability of labour and supply of wire netting, raised it to serious proportions. This was the situation when the Wildlife Section of the Commonwealth Scientific and Industrial Research Organization was formed in 1949, and to it was allotted the task of making a biological study of the rabbit in Australia. This long-term project, it is pointed out by the officer-in-charge of the section, F. N. Ratcliffe, in a recently issued booklet,[2] was considered an essential preliminary to any major progress in dealing with the problem. However, in view of the serious increase in rabbit numbers, it was considered desirable to explore at the outset lines of investigation that offered promise of short-term benefits, even though these might not be far-reaching. A preliminary understanding was sought of the different regional aspects of the problem, and of the potentialities and limitations of available control methods; local and overseas experts were consulted in an attempt to discover and assess ways and means of improving control weapons. The results of the survey form the basis of Ratcliffe's report. This is not an appropriate place to consider the details of rabbit control; those interested can obtain copies of the report from the Secretary, C.S.I.R.O., 314 Albert Street, East Melbourne. It is sufficient to say that a survey of research possibilities in the light of present knowledge shows no line of investigation that seems likely to lead to any revolutionary innovation in methods of rabbit control (other than the use of myxomatosis, which will benefit certain areas only, and of which we shall say more later). However, there appear to be possibilities of development in the fields of fumigation and of poisoning, especially the latter, and suggestions are made for a small-scale experiment in "district control"; this would determine what was involved in, and what could be achieved by, an organized campaign having as its object the complete eradication of rabbits from selected areas.

The feature of rabbit control that has attracted most recent publicity and that is of most medical interest is the use of myxomatosis. Myxomatosis is an infectious disease of rabbits, caused by a virus and characterized by widespread lesions resembling myxomata. Its general manifestations (swollen head, "bunged-up" and discharging eyes, and general "dopeyness") begin to appear on the seventh or eighth day after infection and render the disease unmistakable in the advanced stages. Death usually occurs by the twelfth or thirteenth day at the latest. The disease can be transmitted from affected to healthy rabbits directly and by several Australian mosquitoes. Earlier investigations showed that myxomatosis had very limited powers of natural spread and not a great deal was expected of it. When Ratcliffe's original report was written, the first experiment with myxomatosis was just completed and had been "thoroughly disappointing", as the infection failed to spread effectively even within the warren colonies into which it was introduced. Judgement was reserved, but no great optimism was entertained. However, a footnote to the original report and a supplementary report reveal that myxomatosis has subsequently spread rapidly, through the agency of mosquitoes, first along the Murray River system, then north along the Murrumbidgee, Lachlan and Darling Rivers almost to the Queensland border, and south as far as Benalla. Outbreaks have occurred in South Australia. The mosquitoes responsible for the spread appear to be the dusk-biting *Culex annulirostris* and day-biting species of the genus *Aëdes*. In the most recent report issued by the Commonwealth Scientific and Industrial Research Organization, it is stated that in all those areas in which detailed inspections have been carried out the findings are substantially the same. Active transmission and high mortalities among rabbits have been confined to relatively narrow stretches along the water frontages and to swampy areas where mosquitoes are, or have recently been, prevalent. In some of the old outbreak areas the epidemic shows signs of slowing down, virtual extermination of the rabbit population being achieved in the more favourable spots and little progress being observable elsewhere. It is not anticipated that the disease will become general, and it is specially emphasized that there should be no relaxation of normal control measures by landholders. The disease is not the general solution of the rabbit problem, but it does now appear likely to achieve useful results. Much has yet to be learnt of the disease, and observation of its further progress should be informative. The obtaining of the collaboration of Sir Macfarlane Burnet and of Professor Frank Fenner in the research is a progressive step, and every effort is being made to gain the assistance of interested observers (landholders and so on) on the spot.

One last point requires special mention. It is the question of spread of the disease beyond rabbits and is best answered by quoting from the report:

> It is perhaps advisable to emphasize that the fears being felt and expressed in some quarters lest the infection spread to human beings, stock, and dogs, are quite unfounded. The safety of the virus was established by extensive tests before permission was granted by the health authorities for experiments with it in the field.

There appears therefore to be no danger that myxomatosis will intrude itself into the field of ordinary medical practice, and the medical profession may with interest watch from the sidelines this experiment in what Ratcliffe refers to as "the science of the destructive use of disease".

[1] THE MEDICAL JOURNAL OF AUSTRALIA, February 24, 1951.
[2] "The Rabbit Problem—A Survey of Research Needs and Possibilities", by F. N. Ratcliffe; 1951. Melbourne: Commonwealth Scientific and Industrial Research Organization. 9½" × 7", pp. 17. *Gratis.*

Abstracts from Medical Literature.

BACTERIOLOGY AND IMMUNOLOGY.

Cellular Mechanisms of Antibacterial Defence in Lymph Nodes.

R. O. SMITH AND BARRY WOOD, JUNIOR (*The Journal of Experimental Medicine*, December, 1949), originated a study of cellular mechanisms of antibacterial defence in lymph nodes by investigating the pathogenesis of acute bacterial lymphadenitis. They used white rats inoculated intradermally with small doses of type 1 pneumococci, and killed them at intervals of from five minutes to seventy-two hours later. They removed the skin injected and the popliteal and inguinal lymph nodes and examined them histologically, and they prepared blood cultures from the tail vein. Dosage influenced the rapidity of events, but within five minutes organisms were deep in the lymph nodes; fifteen minutes later acute vascular reaction was apparent and polymorphonuclear leucocytes were invading the sinuses and phagocytizing the cocci. After fifteen hours the macrophage cells were increased, and after twenty-four hours the picture was subsiding. A period of nine to fifteen hours was sufficient for the disappearance of organisms; fibrin coagula were present in some of the lymph channels, and granular debris was found in the mast cells of the lymph sinuses. The leucocytes appeared to come from the capillaries and to act by surface phagocytosis of the non-antibody type. Similar changes followed the injection of streptococci and staphylococci in suitable doses.

Studies on the Life Cycle of Spirochætes.

E. D. DELAMATER, R. H. WIGGALL AND MERLE HAANES (*The Journal of Experimental Medicine*, September, 1950) have recorded studies in the life cycle of the Nichols pathogenic *Treponema pallidum* in the rabbit testis, as seen by phase contrast microscopy, following similar studies on two non-pathogenic strains of the organism occurring in culture and in the embryonated egg. The paper records three means of division in the organism. The commonest is simple transverse fission. The second is by development of a bud or gemma, at any point along the spiral, which enlarges, separates and ultimately liberates a single organism. Thirdly, in material which has been under observation for some time, organisms become paired, and a large cystic structure develops, from which a number of young organisms emerge. There are 67 pictures, both of stained specimens and of living organisms seen by phase contrast, illustrating these descriptions. The studies are being continued.

Viral Inclusions and Their Importance in Diagnosis.

HENRY PINKERTON (*American Journal of Clinical Pathology*, March, 1950) has studied the morphology of viral inclusion bodies and their practical importance in the diagnosis of human disease. He states that they vary in size from 1μ to 20μ, but most often do not exceed the diameter of a red cell. They may be in the cytoplasm near the nucleus or intranuclear. Hæmatoxylin and eosin usually suffice to stain them, but Giemsa's stain is essential in the lymphogranuloma-psittacosis group. There are five diseases in which diagnosis rests upon the presence of the inclusion body: *molluscum contagiosum*, salivary gland virus, Adam's infantile pneumonia, Goodpasture's infantile pneumonia and Hecht's giant-cell pneumonia. Trachoma, herpes-like encephalitis, *lymphogranuloma venereum*, and a few others are greatly clarified by the finding of inclusion bodies, while in a large group, including various forms of encephalitis, measles, dengue fever and the common cold, no inclusion bodies are found. The author discusses specific and non-specific inclusions, the value of a positive finding, and the advisability of deep-freezing portions of autopsy material which can be investigated further if the histological examinations reveal inclusions suggestive of virus infection.

The Relative Stability of Schick and Dick Reactions.

IRWIN S. NEIMAN (*The Journal of Infectious Diseases*, May-June, 1950) has observed the relative stability of the Schick and Dick reactions in a series of medical students in whom the tests were repeated at an interval of one to two years. Changes of reaction in both directions were found, and with both reagents. In the Dick test, 57% reacted in the first test; of the non-reactors, 27% had changed on retest, and of the reactors, 35% had lost their response. In the Schick test, 36% of reactors were found, and on retest 24% had ceased to react. Of the original 237 non-reactors, 19 or 7·6% were "Schick-positive". Of the total number tested, the reaction changed in 14%. The author states that the positive to negative change may be explained by normal immunity mechanisms in a community, but the negative to positive change occurred four times as frequently amongst Dick reactors as amongst Schick reactors, and there was no correlation in the direction of change of reaction in the two tests. The author concludes from an analysis of the reactions that the Dick test may not be epidemiologically as reliable as the Schick test.

Chemotherapy of Experimental Tuberculosis.

A. R. MARTIN AND G. T. STEWART (*The British Journal of Experimental Pathology*, April, 1950) have studied the chemotherapy of experimental tuberculosis in mice. They inoculated mice intravenously with a dose of either 0·75 milligramme or 0·02 milligramme moist weight of human tubercle bacilli strain 905, the large dose producing rapidly fatal, necrotic lesions, the smaller dose proliferative and slower lesions. The therapeutic substances used were para-aminosalicylic acid (40 milligrammes per 20 grammes of mouse, given with the foods), streptomycin (given subcutaneously, 1000 units per 20 grammes of mouse), and a compound known as 8388, having a thiosemicarbazone group (given with food, 10 milligrammes per 20 grammes of mouse per day). Two sets of experiments were arranged, one in which treatment was limited to fourteen days, the other in which it was continuous until death of the animal. Results showed that to assess a therapeutic substance, survival, body weight and pathological assessment of lesions were necessary. Para-aminosalicylic acid alone was effective in the early stages, but the mice died ultimately. Similarly with the compound 8388, the longer the experiment continued, the more notable became the survival of streptomycin-treated animals, although proliferative lesions with many bacilli were still noticeable. True remission or elimination of the infection did not occur.

Serological Types of Corynebacterium Diphtheriæ in Australia.

A. A. FERRIS (*The Journal of Pathology and Bacteriology*, April, 1950) has studied the serological types of *Corynebacterium diphtheriæ* in Australia. His cultures were isolated in both Sydney and Melbourne, usually from a swab inoculated onto tellurite medium, and fermentation reactions and colony characteristics were also recorded; all starch fermenters were recorded as *gravis* strains, non-fermenters with small rough colony as *intermedius*, and all others as *mitis*. Antisera were prepared by immunizing rabbits with a short course of formolized organisms injected intravenously. For the actual test the organisms were emulsified in 5% sodium chloride solution and allowed to stand; periods of thirty minutes usually sufficed to give an emulsion suitable for slide agglutination. Human convalescent serum was also used late in the work and helped to define new types. The results showed that the 794 strains of *Corynebacterium diphtheriæ* examined fell into 14 serological types. Considerable differences of frequency were noted between the two cities, and there was good correlation between virulence and serological type. All subjects of malignant diphtheria of the "bull-neck" clinical picture yielded the same organism, *gravis* type II.

The Effect of Organic Acids on Mammalian Tubercle Bacilli.

R. J. DUBOS (*The Journal of Experimental Medicine*, October, 1950) has studied the effect of organic acids on mammalian tubercle bacilli, while in search of an explanation for the extreme scarcity of organisms in the centre of an active, caseating tuberculous lesion. He states that the diminution in oxygen tension *per se* did not appear to be sufficient, nor did the lowering of pH in the lesion appear to reach inhibitory levels. The accumulation of substances produced in the autolytic breakdown of tissue cells suggested another possible mechanism, and so the author tested cultures of human bovine bacilli in liquid and on solid media with different organic acids at different concentrations. Some of these in very low amount acted as stimulants of bacterial growth and in larger amounts completely inhibited multiplication of the bacilli. The stimulating effect was best seen in media containing whole bovine serum. The viability of the organisms exposed to inhibitory concentrations of acids was not seriously impaired, as the organisms grew out in subculture up to eight days later. It was noted that bacteriostatic activity was greatest in the media most satisfactory for growth. Capric acid was the most active compound tested, but shorter aliphatic acids also inhibited growth. The

author discusses his experiments in relation to the survival and multiplication in inflammatory and caseous areas *in vivo*.

Ascorbic Acid and the Production of Antibody in the Guinea-Pig.

D. A. LONG (*The British Journal of Experimental Pathology*, April, 1950) discusses ascorbic acid and the production of antibody in the guinea-pig. This animal was used because it is dependent upon its diet for the vitamin, and develops symptoms of deficiency if the vitamin is withheld. The response to diphtheria toxoid as a primary and as a secondary stimulus was studied in normal controls and in vitamin *C* deficient animals. Animals were given 0·5 millilitre of alum-precipitated toxoid, and their response to intradermal doses of toxin was measured. Little difference was found between cabbage-fed controls, ascorbic acid-fed controls and vitamin-deficient animals. They were then given a second dose of antigen, and ten days later the antitoxin content of the serum was measured; and here there was a pronounced difference. The two control groups attained fairly high levels (0·68 unit and 0·83 unit per level), while the vitamin-deficient animals could not reach higher than 0·03 unit. The author deduces that vitamin *C* can be assumed to have an obscure influence on the production of antibodies in cells already in a "responsive state", but apparently does not influence the "primary conditioning" by the antigen.

HYGIENE.

Dust Particles in Lung Tissue.

T. BEDFORD AND C. WARNER (*British Journal of Industrial Medicine*, October, 1950), in making a physical study of the dust hazards in south Wales anthracite mines, compared the dust particles in sections of a lung of a man who had died from silicosis after many years' work as a shot-firer in an anthracite mine with the dust particles present in the air in the same mine after ripping shots had been fired in the stall roads. He gives detailed accounts of the size distributions of the particles found. These are separate for particles of coal and particles of minerals other than coal. Particles were examined in the nodules and in the reticulation areas. Of all the particles measured 0·3% to 0·4% were larger than 5μ, and particles as large as 8μ were found in both nodules and reticulation areas. The larger particles in the lungs and in the air in the mine were of coal. Of the non-coal particles 86% to 87% were less than 0·8μ in diameter. The size distributions of the particles in air-borne dust in the mine after firing were similar in type, although the proportion of very small particles was greater in the dust than in the lung tissue. However, when allowance is made for the effects of particle size on alveolar retention, the observed size distributions of the air-borne dust are very close to what would be expected to account for the size distributions of the dust particles found in the lung sections. In the nodules and in the reticulation areas the particle size distributions were very similar. Rough calculations of the effect of partial solution of the dust on particle size distribution suggest that the similarity of the size distributions should not be taken to indicate that no solution occurs after the particles are deposited in the lung tissue.

Vanadium Pentoxide Dust.

SVEN-GÖSTA SJÖBERG (*Acta medica Scandinavica, Supplementum* 238, accompanies Volume CXXXVIII, 1950) reports a clinical and experimental investigation on the effect after inhalation of vanadium pentoxide dust. He describes the use of vanadium in the manufacture of hard steel, as a catalyzer in chemical industries and for other industrial purposes, and states that the inhalation of vanadium pentoxide dust is responsible for an occupational disease which hitherto has passed fairly unnoticed and has not been made the object of much research. Although its effects are of a mild nature and the prognosis is favourable, investigation is important because the substance is already of great and steadily increasing technical importance. He has found that the outstanding clinical feature resulting from its inhalation is an acute and in some cases severe irritation of the respiratory tract, sometimes attended by pneumonia. Allergic skin lesions also occur. The results of animal experiments with the dust agree on all essential points with his clinical observations. Treatment adopted has in most cases consisted in change of work, rest and treatment of symptoms. BAL has been used in two cases, apparently with a satisfactory result. Precautionary measures, in the form of devices for elimination of the dust and protective masks, have brought about a decline in the incidence of the disease. Medical supervision of workers is also important as prophylaxis.

Viability of Escherichia Coli in Sea Water.

R. F. VACCARO, M. BRIGGS, C. CAREY AND B. KETCHUM (*American Journal of Public Health and The Nation's Health*, October, 1951) have investigated the distribution of coliform bacteria in sea water. This investigation is of importance in view of the increasing pollution of harbours, foreshores and tidal rivers. The seasonal changes in the bactericidal activity of sea water were studied throughout a complete year and the effects on sea water of various treatments, such as storage, autoclaving, boiling, adding organic matter, pasteurization and chlorination were determined. The authors state that *Escherichia coli* populations are unable to survive when placed in raw sea water. This bactericidal action of sea water may contribute in an important way to the disappearance of coliform bacteria in tidal waters. There is a pronounced seasonal variation in the survival time of *Escherichia coli* in sea water. They are less viable in the summer months than in the winter. Aging of raw sea water results in an increase in the bactericidal activity of the water. Aging of boiled and autoclaved waters produces little or no change in their bactericidal properties. Sea water sterilized by boiling, autoclaving, pasteurizing, and chlorinating has less bactericidal activity than raw sea water. The addition of organic matter decreases the bactericidal activity of sea water, but has less effect than various methods of sterilization. It is concluded that the most probable cause of the death of *Escherichia coli* populations in sea water is an antibiotic action which requires the presence of the normal marine flora.

School Lunches.

M. L. HATHAWAY, F. MEYER AND S. F. ADELSON (*American Journal of Public Health and The Nation's Health*, September, 1950) investigated a number of factors relating to school lunches. Their nutritive value was determined by analyses. In a pilot study of the nutrition of groups of children, heights and weights were measured, physical examinations for signs of nutritional deficiency were carried out, the hæmoglobin and serum ascorbic acid contents of the blood were estimated, and the dietary patterns of the children and their families were determined. About 700 children were examined. A school lunch was found to consist usually of a main meat course with two vegetables and a sweet, which usually consisted of prepared fruit. The outstanding findings from these studies were as follows: (i) menus within the standard lunch pattern differed considerably in the extent to which they furnished certain important nutrients, and the lunches as served frequently failed to meet one-third of the daily dietary allowances recommended by the National Research Council, particularly for ascorbic acid and thiamine; (ii) blood values for hæmoglobin showed little relationship to school lunch participation, but blood values for ascorbic acid were higher for children receiving a school lunch than for comparable children without the lunch; (iii) children having school lunches had better diets than children not having them, especially with respect to calcium, ascorbic acid, and vitamin *A* values.

Coproporphyrinuria as an Index of Lead Absorption.

W. S. JOHNSON AND N. E. WHITMAN (*Archives of Industrial Hygiene and Occupational Medicine*, August, 1950) present a method of screening urinary specimens for lead content. They give the results of the examination of 294 specimens of urine suspected of containing lead. They state that many investigators have shown that porphyrinuria occurs in a number of pathological conditions, such as congenital porphyria, cirrhosis of the liver, pernicious anæmia, hæmolytic jaundice and poisoning from arsenic, acetanilid, paraldehyde, morphine and lead. The method of de Langen and Berg for the determination of urinary coproporphyrin content was modified to serve as a method for screening lead-urine specimens on the basis of the characteristic red fluorescence of coproporphyrin. Two hundred and ninety-four urine specimens were examined for both coproporphyrin and lead. A table of results is given in which the data are divided into seven groups according to the degree of red fluorescence. Of the entire group of specimens, only two showed no red fluorescence when urinary lead concentrations exceeded 0·15 milligramme per litre. It appears that at the beginning of a lead exposure, porphyrinuria is not established as quickly as an elevation of urinary lead content. The authors consider that with further refinement the method could be used to give a rough quantitative estimate of the amount of lead in a specimen of urine from a person exposed to lead.

Special Article.

(Contributed by Request.)

INFLUENZA, 1950–1951.

THE influenza epidemic now prevailing in northern Europe and America had its probable origin in a localized outbreak of the disease which occurred in Sweden in June, 1950. In November, the disease appeared in Denmark. Later it appeared in Norway and in northern Sweden. By December it had infected the whole of Sweden.

The disease was apparently imported into England (Newcastle area) from the Scandinavian focus. It first spread throughout north England, being for a number of weeks localized to the Tyneside and Merseyside. In the early weeks of January, 1951, it spread south over the whole country. Almost simultaneously the Netherlands, Belgium and the north-west part of Germany were lightly infected.

A second focus of infection, more limited and, to all appearances, independent of the first, was discovered in late December in northern Spain. During January the two foci appeared to join across France.

During 1949 the mainland countries of Europe suffered a much more severe form of influenza than appeared in Britain, and this is advanced as a reason why the death rate in Britain in the 1950-1951 epidemic has been higher than that in Europe. During the latter weeks of January the epidemic declined in Scandinavia and Belgium, but continued to extend through England, Ireland, Switzerland and Spain. It remained widespread in the United Kingdom and Ireland during the early weeks of February, and also appeared in Turkey and Yugoslavia, while clinical cases were reported in Canada. During the middle weeks of February the incidence declined in the north of England, but showed some increase in the rest of the country, while in Canada an increased incidence was reported in the eastern Provinces and in localized communities. No outbreak has been reported from the United States of America, but mild cases of the condition have been reported from some of the South American States.

In nearly all outbreaks a virus "A" has been isolated: this has included the outbreaks in Scandinavia, England and Canada.

Clinically, the disease has been marked by no special diagnostic features in the incubation stage, but by a sudden onset with considerable tracheitis. In a large proportion of cases bronchitis and pneumonia occur; these are fairly common in elderly subjects and in those with predisposing factors. Gastro-enteritis has been present in a number of cases, but the reports of this symptom are variable.

Patients affected have a period of pyrexia of about three days with severe prostration, followed by a protracted period of debility.

In England the death rate has been fairly high among elderly people; over 85% of the deaths have been in the age group over fifty-five years.

In Australia quarantine precautions have been taken to prevent, as far as possible, the introduction of influenza, but in view of the fact that the incubation period might be up to four or five days, while the time of travel by air from infected areas is much less than this, no guarantee can be given that patients with the disease in the incubating stage will not enter Australia. It is felt, however, that if in the early cases those affected are prevented from infecting the Australian population in quantity, the severity of the impact will be lessened, and some immunity will be created to prevent the severe death rate which has been apparent in England.

On all aeroplanes arriving in Australia the passengers and crew are strictly examined for signs of influenza, and should cases be discovered the whole of the aeroplane's complement—that is, passengers and crew—is removed to quarantine stations. Isolation is maintained for five days from the onset of the last case of influenza, while those actually affected are kept in isolation until at least there has been a period of seven days without abnormal temperature. So far three aeroplanes have been quarantined on account of the presence of influenza, and so far as is known, no secondary cases have occurred from these aeroplanes.

A similar watch is kept on vessels arriving from infected areas. To date only one vessel has been ordered into quarantine on account of the existence of influenza on arrival in Australia. Several other vessels have reported cases of influenza during the voyage, but in each case a mild epidemic has occurred, and has died out sufficiently long before actual arrival of the ship in Australia to make unlikely the existence on board of patients with the disease in the incubating stage.

A. J. METCALFE,
Director-General of Health.
Commonwealth Department of Health,
Canberra, A.C.T.

Medical Societies.

THE MEDICAL DEFENCE SOCIETY OF QUEENSLAND.

ANNUAL MEETING.

THE annual meeting of the Medical Defence Society of Queensland was held at British Medical Association House, 225 Wickham Terrace, Brisbane, on Wednesday, February 28, 1951, at 5 p.m., the President, Dr. Neville G. Sutton, in the chair.

Annual Report of Council, 1950.

The annual report of the Council for the year ended December 31, 1950, was presented and adopted. The report is as follows.

The Council has pleasure in presenting the forty-ninth annual report of the Medical Defence Society of Queensland for the year ended December 31, 1950.

Membership.

The society has now a membership of 547, as against 542 last year. This figure includes 13 members who are overseas temporarily. During the year 33 new members were elected. Our losses were: deceased 7, struck off owing to default in payment of subscription 4, resigned or left the State 17.

Obituary.

It is with much regret that we record the deaths of the following members: Dr. James Brown, Dr. P. F. Hyndes, Dr. P. J. Kerwin, Dr. A. M. Langan, Dr. Edward Mansfield, Dr. F. G. Meade, Dr. F. C. Wooster.

Office Bearers and Council, 1950.

The following office bearers were unanimously reelected by the Council: *President,* Dr. Neville G. Sutton; *Vice-President,* Dr. G. W. Macartney; *Honorary Treasurer,* Dr. T. V. Stubbs Brown; *Honorary Secretary,* Dr. R. G. Quinn; *Councillors,* Dr. Gavin H. Cameron, Dr. H. W. Horn, Dr. F. W. R. Lukin, Dr. H. Masel, Dr. Athol Quayle, Dr. F. Garrett Scoles, Dr. J. G. Wagner.

Dr. Neville G. Sutton, Dr. T. V. Stubbs Brown and Dr. Gavin H. Cameron, who retired in conformity with the by-laws, were reelected.

Medico-Legal.

The society has had another year free from litigation.

Two cases are still pending, but it is considered unlikely that any further action will develop.

Legal expenses were paid to a member who had been advised to be represented at a coroner's inquest.

No action has yet been taken by the British Medical Association to investigate the position in regard to the assessment by juries of damages against doctors and other professional bodies.

Proposed Australia-Wide Medical Defence Organization.

A meeting of representatives of State medical defence organizations was held in Brisbane on May 29, 1950, at which the following were represented: *New South Wales,* Dr. B. T. Edye and Dr. H. R. R. Grieve; *Victoria,* Dr. Robert Southby and Dr. Leonard Ball; *Queensland,* Dr. Neville G. Sutton; *South Australia,* Dr. C. O. F. Rieger; *Western Australia,* Dr. B. C. Cohen; *Tasmania,* Dr. J. L. Grove.

After discussion the general feeling of the meeting was not disposed to favour the formation of a federal body. It was considered, however, that good would result from the interchange of views, a practice which might be repeated in future.

It was stated that the development of the schemes in operation in Queensland and Western Australia would be followed with interest.

The suggestion was made that the larger States might provide cover for medical practitioners of the smaller States.

No formal motions were put before the meeting.

Indemnity Insurance: Medical Protection Society, Limited.

The arrangement between the two societies is still functioning satisfactorily as regards indemnity insurance of members of the Medical Defence Society of Queensland. During the year a further number of members have taken the opportunity of increasing their cover.

Retired and Deceased Members.—(i) Approval has been given to the compounding of two years' subscription for life membership for retired members at the scale of indemnity on which their final payment is based. (ii) The personal representatives of a deceased member who was a member of the Medical Defence Society of Queensland shall be indemnified for an amount equivalent to the indemnity to which the member was entitled at the time of his death in respect of proceedings taken against his estate on the grounds of some alleged act of negligence or omission arising from his professional work during the period he was a member of the society.

Any member admitted to life membership is required to notify the society, at least once in every two years, of his present address. Failure to do so for a period of two years will result in the termination of his membership. The rules of the society in regard to life membership of the Medical Protection Society, Limited, were circulated to members of the Medical Defence Society of Queensland through the Queensland Branch of the British Medical Association.

Locum Tenens.—We have received information to the effect that the society indemnifies a member in respect of any actions which may be taken against him arising from the professional acts of his *locum tenens*, but does not indemnify the *locum tenens*, should he be joined in any such proceedings, unless he is a member of the society. This benefit is given without payment of any additional subscription by a member. It is, however, obligatory on the member that he should exercise due care and discretion in appointing a *locum tenens*—to ascertain that he is a registered medical practitioner and obtain suitable references.

Finance.

The net surplus of the society for the year ending December 31, 1950, was £328 1s. 8d.

Items of income and expenditure are as follows:

	£ s. d.	£ s. d.
Entrance fees and membership subscriptions		320 11 3
Interest on Commonwealth Stock and Savings Bank		244 2 10
		564 14 1
Legal expenses	29 8 0	
Secretary's salary and clerical assistance	154 0 0	
Postage, duty stamps, exchange and sundries	18 11 0	
Printing and stationery	11 10 9	
Office rent	5 0 0	
Audit and accountancy (two years)	17 17 0	
Depreciation	5 8	
		236 12 5
		£328 1 8

Indemnity insurance remittances amounted to £2481 11s. 4d.

A total of £7163 18s. is invested in Commonwealth Government Loan, and bank balances and cash amount to £890 16s. 1d.

The total assets of the society as at December 31, 1950, amounted to £8060 0s. 11d.

All contingencies have been provided for, with the exception of Federal income tax and further tax on undistributed income to be assessed on the taxable income of the current year.

NEVILLE G. SUTTON,
President.

Balance Sheet and Financial Statement.

The balance sheet and financial statement for the year ended December 31, 1950, was adopted.

Election of Councillors for 1951.

The following members, who had retired in accordance with the by-laws, were reelected: Dr. H. W. Horn, Dr. F. W. R. Lukin and Dr. J. G. Wagner.

Election of Auditors.

Messrs. R. G. Groom and Company, Chartered Accountants (Australia), were reelected auditors for the ensuing year at a remuneration to be fixed.

General Business.

Damages Assessed Against Doctors.

The matter of damages assessed against doctors was again discussed, and it was resolved that no further action be taken.

Correspondence.

RECENT ADVANCES IN CANCER RESEARCH.

SIR: It is seldom if ever possible to settle problems in science by mere verbal argument. The history of our knowledge concerning filtrable tumours of the domestic hen—the tumours usually referred to as the Rous sarcoma—is a perfect example of this well-known truth. The first of these tumours, a delicate spindle-celled sarcoma, came under observation in 1910 and was regarded by members of the Pathological Society of New York as a beautiful example of an avian neoplasm. In 1911 Rous demonstrated that the tumour could be propagated by means of cell-free filtrates; the tumours were at once placed in a separate category and thenceforth regarded as a disease *sui generis*. In England E. F. Bashford coined a new name for a tumour which is transmissible by cell-free extracts without living cells, namely, a "Rousoma". In Germany, Aschoff, possibly the last of a succession of great German pathologists, formed a team of workers bent on proving that the active filtrates contain minute perfect cells, smaller than 0·2μ, capable of passing through a Berkefeld filter candle, and when injected, multiplying within the tissues to form a daughter tumour. After several years of close concentrated hard work failure was their lot. Several years after Rous had proved that filtrates of true avian neoplasms contain an agent with virus-like properties capable of starting tumours afresh, multiplying *pari passu* with the growth of the new tumour, Murphy, Rous and their colleagues demonstrated that tumour tissue dried *in vacuo* over sulphuric acid (or phosphorus pentoxide) retains this power of tumour propagation; this was regarded as a final and complete proof that the filtrable tumours are maintained as tumours by the presence and action of an intracellular virus. But even as recently as the early nineteen thirties a Japanese pathologist published a paper in GANN which claimed to prove that cells which have been dried in the approved fashion contain a small, sometimes a large, percentage of living viable cells and that the tumour which forms after the injection of the dried tissue is derived from these cells. The results were repudiated and the claims withdrawn within three or four months of publication.

This slightly complex but long-drawn-out history of the filtrable avian sarcomata will almost certainly be followed more or less exactly by the history of filtrable mammalian cancers. Could you expect us, sir, to reply in detail to the comments in your leading article of February 10? Or, more pertinently, sir, could we expect you to afford us sufficient space to reply adequately?

Perhaps you will be good enough to give us two more paragraphs. The deep fixed belief—prejudice if you will—that the only way to keep tumours alive is to transplant live cells in appropriate animals and to continue doing this at intervals governed by the properties and the state of the tumour is due to the experiments carried out on a large—perhaps huge would be a permissible word—scale by Bashford, Haaland, Cramer and Russell in the early part of this century. The conclusion drawn from them was briefly that the tumours which are maintained in laboratories for experimental purposes are composed entirely of lineal descendants of the original tumour cells which arose in an animal, for example, a mouse, months or years before, and that it is merely foolish or a waste of time to attempt to extract a continuing cause from any tumour. This was, of course, before the work of Rous and his colleagues had been published. But the effects of the

propaganda of Bashford, Murray, Ewing *et alii* continue and will continue undoubtedly until a practical consequence of the discovery, that the whole basis of cancer research (*cancérologie* as it is called on the continent of Europe) is an error, emerges from the new knowledge.

Finally, with regard to the work of Passey *et alii*, the following comments appear to us to be fair. In the first place, is there anything in Professor Passey's work which, in the light of the foregoing remarks, is against the possibility that the unit of transmission of cancer is something smaller than the cell itself and multiple within it? We are grateful for him for having so meticulously confirmed our results with dried tumour material, and he would appear to differ from us merely in his interpretation. He states that some, but not all, of the cells may survive freezing, but he does not explain why, if some are killed by cold, the frozen material increases in activity with increase in length of freezing time. Also, since rapid freezing is known to be a good histological fixative, we fail to see how to tell a live frozen cell from a dead frozen cell. Secondly, why is drying only successful in extreme cold? One would expect a mammalian cell to survive better if dried above the eutectic point (at which temperature, of course, the water and salt separate out, and the cell is, as it were, pickled in brine before becoming entirely solid) than if dried below it. Thirdly, it is well known, and we have stated it ourselves in previous publications, that the sediment alone in sand-ground centrifuged frozen material is active, and that this consists of naked nuclei and not intact cells. There is therefore something inside the nucleus which transmits the tumour. With the behaviour of other viruses (for example, phage encysted within the bodies of bacteria) this at least suggests an intranuclear unit of transmission rather than a cellular one. Finally (or we shall try your patience, sir, too much), in view of recent advances in knowledge of the behaviour of other viruses, is there a greater likelihood of the survival of an intact complete mammalian cell at the temperature of liquid air or of the survival of an intranuclear virus?

Yours, etc.,

W. E. GYE.
IDA MANN.

56 Hobbs Avenue,
Nedlands,
Western Australia.

March 3, 1951.

THE GROCERS' COMPANY SCHOLARSHIP.

SIR: In a recent issue of your journal (February 10, 1951, page 241) a letter from the Commonwealth Director-General of Health appeared under a heading which arrested my attention; it told of a generous offer made by the Grocers' Company to provide a scholarship for medical research. I imagined that the brethren at Grocers' Hall, London, must have remembered with kindly feeling the erstwhile association of their ancient guild with that lowly branch of the medical profession, the apothecaries. At all events, I thought it might be of interest to some of your readers to have part of this chapter in our struggle for existence recalled.

From the Middle Ages to the end of the eighteenth century the apothecaries were the recognized drug-sellers in Great Britain, and many of them were engaged in active medical practice as well. A lucrative side-line of the grocers' trade also comprised the importation and retail sale of drugs and medical supplies. If the business rivalry between the two was at first friendly, it soon changed to an interminable and bitter antagonism after Edward III, in 1345, allowed the grocers to become a corporate body by letters patent, which placed the apothecaries under their jurisdiction for regulation and control. Now the grocers were given power to examine the drugs and medical wares of the apothecaries before they could be sold in their shops, and in the event of any irregularity being detected by the company, the offending apothecary was punished either by fine or imprisonment.

During the sixteenth and seventeenth centuries these humble general practitioners of medicine found themselves in frequent conflict with the austere physicians, who, in 1511, had persuaded Henry VIII to curb their unlicensed professional activities by Act of Parliament. However, it was not until 1616 that the apothecaries were able to break away finally from their old antagonists, the grocers, when they were given a charter by James I enabling them to unite in a separate city guild under the title "Society of Apothecaries". The grocers were greatly incensed at this unseemly move for independence, and they petitioned to king to have it revoked. But James I merely retorted that the grocers were ordinary merchants, while the business of the apothecary was a mystery, and for this reason alone he thought they were entitled to their own corporation.

The subsequent history of these ancient guilds hardly concerns us here. Suffice it to say that the apothecaries gained a lasting legal victory over the physicians in an appeal which came before the House of Lords in 1703; that they were given legal status as medical practitioners in the *Apothecaries Act* of 1815, by which time the chemists and druggists had taken over most of their shops; and that any modern householder can still order aperients, tonics, pills, antacid powders, headache tablets and vitamin preparations from the local grocer.

Yours, etc.,

K. MACARTHUR BROWN.

Parramatta,
February 23, 1951.

MEDICINE AND ATOMIC WARFARE.

SIR: May I refer to a possible source of defeatism in your commendably realistic editorial "Medicine and Atomic Warfare" (THE MEDICAL JOURNAL OF AUSTRALIA, February 24, 1951)?

You have suggested that "international inspection and control . . . could be . . . only in a world devoid of suspicion and lust for power. . . . In no single country has this ideal state made its appearance, and until that happens it is foolish to think of its manifestation in the international sphere".

If this is so we are in a poor way indeed, for then mankind will never escape the threat of bigger and bigger wars, and furthermore most attempts at supranational federations would seem to be doomed. History seems to suggest the more optimistic hope that federations between groups with diverse interests can be "hammered out", in spite of suspicion and lust for power, when the demand for federation becomes vocal and active among the citizens affected. Bloodshed is, of course, often needed to effect federations, or to protect them, as it was in the American Civil War and may well be in World Wars III, IV and so on.

May I commend to those who share your ideals of extending the brotherhood of man that they should contact and join the World Movement for World Federal Government? This movement has the support of many champions of health (including Lord Boyd Orr, world president of the movement, and Dr. E. S. Morris, New South Wales Director-General of Public Health) and of leaders in every sphere, from Professor Einstein to Pandit Nahru and (in principle) Winston Churchill.

The essential weaknesses of the "alliance" principle in bodies like the League and the United Nations Organization are being clarified by these people. They are even now arranging a world conference to formulate their proposals. I suggest that Australians have progressed far since the days of Governor Phillip only because they, similarly, did not wait for national brotherhood before demanding national law and order.

Full details of the movement are available from G.P.O. Box 3904TT, Sydney.

Yours, etc.,

DOUGLAS EVERINGHAM.

Mental Hospital,
Gladesville,

February 24, 1951.

CASES FOR DIAGNOSIS.

SIR: I read with interest Dr. F. A. Rodway's letter entitled "Cases for Diagnosis" in your issue of February 17, 1951. He describes a local epidemic of six afebrile cases of puffiness of the eyelids, sometimes severe enough to close the palpebral fissure, associated with mild conjunctivitis.

From time to time we oto-rhino-laryngologists have patients referred with similar symptoms diagnosed as acute frontal sinusitis or ethmoiditis, whereas the real cause is overlooked, Morax-Axenfeld infection of the conjunctival sac. Not infrequently there may be headaches and nasal or pharyngeal symptoms. The interesting point in the type I refer to is the mildness of the signs of infection of the conjunctival sac compared with the marked degree of the surrounding œdema. Practically always, however, one can detect a horizontal band of congestion of the ocular con-

junctiva at the level of the closed palpebral fissure; hence the other name of the disease, namely, angular conjunctivitis.

Though I think it unlikely that Dr. Rodway should encounter six of these unusual types of angular conjunctivitis without seeing at the same time at least some of the more usual types with obvious infection of the conjunctival sac and little or no œdema, I make the suggestion both for his information and to ventilate a not-well-enough-known cause of palpebral and frontal swellings. Should he encounter any more such cases I suggest an examination for the tell-tale angular congestion and a pathological examination for the diplo-bacillus in question.

Yours, etc.,

A. B. K. WATKINS.

Commercial Bank Chambers,
Bolton Street,
Newcastle,
New South Wales.
February 20, 1951.

SIR: In reply to Dr. F. A. Rodway's letter, "Cases for Diagnosis" (THE MEDICAL JOURNAL OF AUSTRALIA, February 17, 1951), in my opinion all five cases were caused by a poisonous fly bite. The monocular incidence and the recovery within a few days are typical of this lesion. In other words, I think the condition is a reaction, by œdema, to a chemical protoplasmic poison.

When in practice many years ago in the Maitland district I saw many such cases during the summer months. The œdema is worse on awakening in the morning, subsides somewhat during the day, and partially relapses the next morning; from three to six days is the usual period before complete subsidence.

On one occasion my patient actually felt the bite, captured the fly and brought it to me. Unfortunately I did not have the fly identified, but it was darker and smaller than the ordinary house fly.

It is interesting that the one case in which the œdema spread to the other side was the one which did not at once receive sulphadiazine, but I think the explanation is that this patient received a larger dose of formic acid; it seems to me unlikely that a sulphonamide would have any inhibitory effect upon the body's reaction to a chemical substance.

Yours, etc.,

ARTHUR D'OMBRAIN.

135 Macquarie Street,
Sydney,
February 22, 1951.

AN APPRECIATION.

SIR: Recently the Council received advice from the London office that Dr. C. R. Hodgson had completed fifty years' membership of the Association and was accordingly entitled to continue his membership without payment of further subscription.

The congratulations of the Council were extended to Dr. Hodgson, and in reply the Council received a letter from him, copy of which I enclose. The Council would be pleased if you could see your way clear to publish this letter in THE MEDICAL JOURNAL OF AUSTRALIA.

Yours, etc.,

J. G. HUNTER,
Medical Secretary, New South Wales Branch of the British Medical Association.

135 Macquarie Street,
Sydney,
February 23, 1951.

[COPY.]

Dear Dr. Hunter,

I appreciate very much the Council's letter of the 18th inst. and many courtesies in the past, and I should like to pay tribute to the high standard of the *British Medical Journal* and THE MEDICAL JOURNAL OF AUSTRALIA.

I left The King's School when my parents returned to England and continued at school there, and qualified at London University from Guy's Hospital. One of my first reactions after passing the M.D. was to join the British Medical Association, and another was to join up for two years' service in the Boer War, whose veterans I still look after, as well as Light Horse comrades in the 1914 war.

Your letter makes me realize that I qualified in the last century, so may I celebrate it by enclosing a small cheque for the Medical Benevolent Fund, of which I am a Life Member.

Yours sincerely,

(Signed) C. R. HODGSON.

35 Wolseley Road,
Point Piper,
Sydney.
December 20, 1950.

ACUTE ANTERIOR POLIOMYELITIS.

SIR: In my own practice may I support the observations of J. Forbes Mackenzie (THE MEDICAL JOURNAL OF AUSTRALIA, March 3, 1951). Over a period of years I have observed that, preceding every major epidemic of poliomyelitis, there has been an outbreak of *herpes zoster*, chiefly confined to adults and especially those in the aged group—this apart from any associated epidemic of chickenpox.

Repeatedly I have drawn the attention of many of my fellow practitioners to this occurrence, coincidental as it may be. Nevertheless I feel that virus infection can be likened to that of chain reaction, with its associated mutative changes. "Contramine" as a rule has given very good results, whole liver good and bad results. Autogenous whole blood on occasions has worked like a charm. All these were given intramuscularly. I have found none of these preparations to be absolutely specific. Failure with one means try with another. Then they are not always successful. In some of the aged, why the degree of debilitation with the appearance of minimal rash? Yet there will occur in the same age group an extensive rash with a minimum constitutional upset.

May I raise this point—what is the relationship of salt metabolism to diseases of the nervous system, for example, virus disease? Observe the general improvement in health in these debilitated people by increasing their salt intake, especially if combined with an adrenal cortical preparation. I have noted this previously in your journal in relation to pink disease. Might I ask, what is a virus? Is it a crystalloid? If so, then it is a molecule. There are special types of molecules. I see no reason as to why a virus could not conform to a living molecule. More so as it is obligate. Then, too, the possibility that a virus may be derived from a distorted protein. This then in turn opens up the field of solar radiation. How do they work? Is it through the enzyme system of the host?

To make progress in the study of virology, I am of the opinion that it must be removed from the atmosphere of bacteriology and be more closely linked to that of biophysics.

Yours, etc.,

KENNETH ADDISON.

Waverley,
New South Wales,
March 5, 1951.

SIR: I read with great interest the letter of Dr. J. Forbes Mackenzie, published in your issue of the journal of March 3, 1951. In support of his theory I would like you to publish the following details of a case now under my care.

I saw Mrs. C., aged fifty-four years, on February 22, 1951, when she complained of "a pain in my head and a tight pain in the lower part of my back". She was mentally alert and very definite that this disability was quite unlike the usual aches and pains of a harassed housewife. In addition she complained of "a quivering sensation almost like pins and needles" in all four limbs. A pyrexia of 100° F. was present, and the pulse rate was 100 per minute. There was pain on flexing the neck, and the stiffness of the spine was very definite, but with no neck retraction or opisthotonos. No other neurological signs were present. Next day there was some slight improvement, and lumbar puncture performed by Dr. A. Hogen revealed slightly turbid cerebro-spinal fluid with 900 cells (90% lymphocytes) per cubic millimetre and 200 milligrammes of protein *per centum*. During the performance of the lumbar puncture a small patch of painless erythema was noticed on the left side of the interscapular area, which by the following day had developed into a frank *herpes zoster* rash in the area supplied by the third left thoracic segment of the cord. Curiously there was very little pain and hyperæsthesia associated with the rash, but its appearance was typical and classical. With the exception of the rash there was a marked improvement in the symptoms, and one week after

the onset all spinal movements were free and the vesicles healing. Because of the mildness of the symptoms, no specific therapy was employed, and a proprietary preparation of aspirin and codein was adequate. Prior to the appearance of the rash and with the confirmatory evidence of the cerebro-spinal fluid findings, the diagnosis was that of acute poliomyelitis, but revised to *herpes zoster* the following day.

Dr. Bradfield, in THE MEDICAL JOURNAL OF AUSTRALIA of February 17, 1951, mentions *herpes zoster* as one of the conditions which might be confused with acute poliomyelitis, but he does not make it clear if the confusion may arise because of pain in the affected dermatome or because of more central involvement. That possible confusion between these two diseases can be due to spinal cord involvement is shown by the cerebro-spinal fluid findings, and furthermore the striking similarity of biochemical and clinical signs must lend support to the theory of Dr. Forbes Mackenzie.

Yours, etc.,
M. A. O'HALLORAN.

111 Gerrale Street,
Cronulla,
New South Wales.
March 2, 1951.

ECONOMY OF MEDICAL SUPPLIES.

SIR: The expenditure within the community of certain therapeutic measures which may be restricted in a national emergency is causing some concern, and it is considered reasonable that attention should be drawn to this among the medical schools throughout Australia. Medical students and resident medical officers during their training could well be impressed with the importance of economy in the proper use of dressings, antibiotic drugs, blood and its derivatives, special anæsthetics and X-ray films. Economy is compatible with efficiency—waste is not. This recommendation is made in national interests, as it may be expected that any emergency medical services of the future will be staffed largely from the present-day medical students and resident medical officers.

Yours, etc.,
F. KINGSLEY NORRIS,
Major-General, Director-General of Medical Services.

Army Headquarters,
Victoria Barracks,
Melbourne, S.C.1.
March 2, 1951.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

GENERAL REVISION COURSE, 1951.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that the annual general revision course will be held in Sydney for two weeks beginning April 2, 1951, under the supervision of Dr. R. L. Harris. Fees for attendance will be as follows: full course, £6 6s.; mornings or afternoons only, £3 3s.; one week only, £3 3s. Early application, enclosing remittance, should be made to the Course Secretary, the Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 5238, BW 7483. Telegraphic address: "Postgrad, Sydney." Candidates may submit in writing questions for the panel discussions; these must be received at the office not later than Monday, March 26, 1951.

Programme.

The programme will be as follows.

Monday, April 2.—In the British Medical Association Council Room, 135 Macquarie Street, Sydney: 9.15 a.m., registration and opening of course, Colonel A. M. McIntosh; 10 a.m., "Hypertension", Dr. C. G. McDonald; 11.15 a.m., "Management of Acute Retention of Urine", Dr. M. F. Nicholls, Senior Surgeon, Saint George's Hospital, London (guest lecturer). At the Royal Alexandra Hospital for Children, Bridge Road, Camperdown: 2.15 p.m., pædiatric problems (medical, Professor Lorimer Dods; surgical, Dr. J. Steigrad).

Tuesday, April 3.—In the British Medical Association Council Room: 9.15 a.m., "Cardiac Emergencies" Dr. John H. Halliday; 10.45 a.m., "Skin in General Practice", Dr. J. Witton Flynn; 11.45 a.m., "Tuberculosis in General Practice", Dr. Bruce White; 2.15 p.m., "Anæsthesia in General Practice", Dr. W. I. T. Hotten; 3.45 p.m., panel discussion, "Psychosomatic Medicine", Dr. T. M. Greenaway and Dr. Cedric Swanton.

Wednesday, April 4.—In the British Medical Association Council Room: 9.15 a.m., "Albuminuria in Pregnancy", Dr. Richmond Jeremy; 10.45 a.m., panel discussion by obstetricians, "Use and Abuse of Cæsarean Section", Professor Bruce T. Mayes, Dr. T. H. Small and Dr. W. D. Cunningham; 2.15 p.m., "Common Diseases of the Rectum and Anus", Dr. V. M. Coppleson; 3.45 p.m., "Epilepsy and its Management", Dr. Eric Susman.

Thursday, April 5.—In the British Medical Association Council Room: 9.15 a.m., "Infectious Diseases", Dr. N. J. Symington; 10.45 a.m., "Infant Feeding", Dr. Kathleen Winning; 11.45 a.m., "Treatment of Migraine", Dr. Kenneth B. Noad; 2 p.m., "Recent Advances in Cardiology", Dr. J. Kempson Maddox; 3.45 p.m., panel discussion, "Problems in General Practice", Dr. W. F. Simmons, Dr. A. C. Thomas and Dr. Gordon Lowe.

Friday, April 6.—In the British Medical Association Council Room: 9.15 a.m., "Anæmias and Their Treatment", Dr. F. A. E. Lawes; 10.45 a.m., "Everyday Thyreoid Problems", Dr. F. F. Rundle; 11.45 a.m., "Painful Shoulders", Dr. Selwyn G. Nelson. At Royal Prince Alfred Hospital, Missenden Road, Camperdown: 1.30 p.m., clinical afternoon.

Monday, April 9.—In the British Medical Association Council Room: 9.15 a.m., "Infections of the Hands", Dr. Eric A. Hedberg; 10.45 a.m., "Ear, Nose and Throat in General Practice", Dr. Ashleigh O. Davy; 11.45 a.m., "Management of Head Injuries", Dr. Rex A. Money. At the Robert H. Todd Assembly Hall: 2.15 p.m., seminar, "Poliomyelitis", Dr. Laurence Hughes and Dr. D. G. Hamilton; 3.45 p.m., seminar, "Investigation of Dyspepsia", Dr. V. J. Kinsella, Dr. F. W. Niesche and Dr. J. E. Sherwood.

Tuesday, April 10.—In the British Medical Association Council Room: 9.15 a.m., "Surgical Treatment of Essential Hypertension", Dr. I. Douglas Miller; 10.45 a.m., "Coronary Heart Disease", Dr. E. H. Stokes; 11.45 a.m., "Snakes and Snake Venom", Mr. J. R. Kinghorn.

Wednesday, April 11.—In the British Medical Association Council Room: 9.15 a.m., seminar, "Jaundice", Dr. Bruce Hall and Dr. Norman Wyndham; 10.45 a.m., seminar, "The New Drugs and Antibiotics, including ACTH and Cortisone", Professor H. K. Ward, Dr. A. W. Morrow and Dr. John Loewenthal. At Broughton Hall Psychiatric Clinic, Wharf Road, Leichhardt: 2.15 p.m., psychiatric demonstration, "Depressive States".

Thursday, April 12.—In the British Medical Association Council Room: 9.15 a.m., "Management of Pleural Effusion", Dr. A. J. Collins; 10.45 a.m., "Orthopædic Problems in General Practice", Dr. McC. Callow; 11.45 a.m., "Diabetes in General Practice", Dr. W. Wilson Ingram; 2.15 p.m., "Ophthalmology in General Practice", Dr. John Antill Pockley; 3.45 p.m., seminar, "Gynæcology in General Practice", Dr. F. A. Maguire, Dr. C. V. Salisbury, Dr. S. Devenish Meares, Dr. J. Cameron Loxton, Dr. M. Britnell Fraser and Dr. Malcolm Stening.

Friday, April 13.—In the British Medical Association Council Room: 9.15 a.m., "Hospitals and Hospital Administration", Dr. A. B. Lilley; 10.45 a.m., "Management of Gall-Bladder Disease", Dr. S. H. Lovell; 2.15 p.m., film session; 3.45 p.m., question session, Dr. Raymond Green, Dr. Bruce Williams, Dr. Frank Mills and Dr. Frank Ritchie.

The Royal College of Obstetricians and Gynaecologists.

VICTORIAN STATE COMMITTEE.

UNDER the auspices of the Victorian State Committee of the Royal College of Obstetricians and Gynæcologists, Sir William Gilliatt, Past President of the College, will deliver two lectures in the College Hall, 122 Flinders Street, Melbourne, as follows:

Thursday, April 26, 1951, at 8.15 p.m.: "Practical Points in Obstetrics."

Tuesday, May 1, 1951, at 8.15 p.m.: "Practical Points in Gynæcology."

The lectures will be suitable for general practitioners. The fee for two lectures is one guinea, or 10s. 6d. per single lecture.

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 14, of March 1, 1951.

NAVAL FORCES OF THE COMMONWEALTH.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Promotions.—Surgeon Lieutenant (for short service) Joseph Lee Watson is promoted to the rank of Surgeon Lieutenant-Commander (for short service), dated 2nd January, 1950.

Ante-dating Seniority.—The seniority of Joseph Lee Watson as Surgeon Lieutenant (for short service) is antedated to 2nd January, 1946.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Volunteer Reserve.

Promotion.—Surgeon Lieutenant Jack Ramsay Collie is promoted to the rank of Surgeon Lieutenant-Commander, dated 20th November, 1950.

AUSTRALIAN MILITARY FORCES.

Royal Australian Army Medical Corps (Medical).

NX506297 Captain (Temporary Lieutenant-Colonel) J. Prior relinquishes the temporary rank of Lieutenant-Colonel, 29th December, 1950.

Citizen Military Forces.

Northern Command: First Military District.

Royal Australian Army Medical Corps (Medical).—1/39091 Captain (provisionally) D. J. Chapman relinquishes the provisional rank of Captain and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (1st Military District) with the honorary rank of Captain, 6th November, 1950.

Central Command: Fourth Military District.

Royal Australian Army Medical Corps (Medical).—4/31956 Captain L. C. Lum is seconded whilst undergoing post-graduate studies in the United Kingdom, 23rd October, 1950. 4/35229 Honorary Captain D. A. Simpson is appointed from the Reserve of Officers, and to be Captain (provisionally), 30th November, 1950. The provisional ranks of the following officers are confirmed: Majors 4/31903 C. M. Gurner and 4/31904 P. S. Eyles.

Tasmania Command: Sixth Military District.

Royal Australian Army Medical Corps (Medical): To be Captain (provisionally), 4th December, 1950.—6/9210 Athol Charles Donne Corney.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps (Medical).

To be Honorary Captains, 19th December, 1950.—Robert Barnes and Wolfram Immanuel Selth.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED FEBRUARY 17, 1951.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.[2]	Western Australia.	Tasmania.	Northern Territory.[3]	Australian Capital Territory.	Australia.[3]
Ankylostomiasis	•	..	7	..	..	..	..	..	7
Anthrax	•	..	..	..	..	..	..	..	..
Beriberi	•	•	•	•	•	•	..	•	..
Bilharziasis	•	..	..	..	..	..	..	..	..
Cerebro-spinal Meningitis	1	3	..	..	..	..	..	..	4
Cholera	..	..	..	..	..	..	..	..	..
Coastal Fever(*a*)	•	•	..	•	•	•	•	•	..
Dengue	•	•	•	•	•	•	..	•	..
Diarrhœa (Infantile)	..	..	2(2)	..	6(4)	..	..	..	8
Diphtheria	8(5)	4(4)	7(5)	..	14(14)	..	..	..	33
Dysentery (Amœbic)	•	..	..	..	1	..	..	..	1
Dysentery (Bacillary)	•	3(2)	..	..	4(4)	..	•	4(4)	11
Encephalitis Lethargica	..	..	..	..	..	..	..	..	..
Erysipelas	•	•	•	..	•	•	..	..	..
Filariasis	•	..	..	..	..	..	..	..	..
Helminthiasis	•	..	•	•	•	•	•	•	..
Hydatid	•	..	•	•	..	•	•	..	..
Influenza	•	•	•	..	•	•	..	•	..
Lead Poisoning	•	•	..	•	•	•	•	•	..
Leprosy	..	..	1(1)	..	1	..	..	..	2
Malaria(*b*)	•	..	..	..	..	..	..	..	..
Measles	•	•	•	13(1)	•	•	..	..	13
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	58(24)	12(2)	32(2)	25(17)	6(2)	12(6)	•	•	145
Psittacosis	•	..	•	..	•	•	..	..	..
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella(*c*)	•	•	•	..	2(1)	•	..	..	2
Scarlet Fever	11(7)	10(5)	12(8)	3(2)	7(7)	..	..	..	43
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	•	..	..	•	•	•	•	•	..
Trachoma	•	..	..	•	•	•	•	•	..
Tuberculosis(*d*)	14(8)	31(21)	29(15)	6(3)	3(2)	3	..	..	86
Typhoid Fever(*e*)	1(1)	..	..	..	2(2)	1	..	..	4
Typhus (Endemic)(*f*)	..	..	1	1(1)	..	..	..	..	2
Undulant Fever	..	..	..	..	..	..	..	..	..
Weil's Disease(*g*)	•	•	6(1)	•	•	•	•	•	6
Whooping Cough	•	•	•	1(1)	•	•	..	..	1
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 37, 1946–1947. Figures in parentheses are those for the metropolitan area.

[2] Figures not available.

[3] Figures incomplete owing to absence of returns from the Northern Territory.

• Not notifiable.

(*a*) Includes Mossman and Sarina fevers. (*b*) Mainly relapses among servicemen infected overseas. (*c*) Notifiable disease in Queensland in females aged over fourteen years. (*d*) Includes all forms. (*e*) Includes enteric fever, paratyphoid fevers and other Salmonella infections. (*f*) Includes scrub, murine and tick typhus. (*g*) Includes leptospiroses, Weil's and para-Weil's disease.

ROYAL AUSTRALIAN AIR FORCE.

Active Citizen Air Force.

The following officers are transferred to the Reserve, 1st October, 1950:

Medical Branch.—Temporary Group Captains G. C. Willcocks, O.B.E., M.C. (1161), H. R. G. Poate, M.V.O. (1162), A. H. Baldwin (264907), C. H. C. Searby, K.H.S. (2041), C. H. Hembrow (1184), N. E. H. Box (251172), C. G. H. Blakemore (1180); Temporary Wing Commanders R. L. T. Grant (1185), I. B. Jose (1851), I. G. McLean (251178), J. G. Brown (251457), E. O. Marks (1166), L. B. Cox (1179), D. R. Gawler (1183), R. M. Glynn (1850), A. L. Tostevin (1883), L. E. Hurley (2075), J. O'Sullivan (2076), H. W. Savige (2077), N. G. Sutton (2078), A. J. H. Stobo (2100), J. J. W. Flynn, M.C. (261268), T. A. Travers (1269), E. M. Fisher, M.C. (2056), N. M. Cuthbert, M.C. (1465), W. C. T. Upton (3455), B. W. B. Riley (264370), H. S. Lucraft (6376), W. N. Little (6410), W. M. Lemmon (251284), J. A. McGeorge (7271), B. T. Mayes, M.V.O. (264848), F. V. Munro (261278), S. M. L. Dunstone (281654), A. R. Robinson (263475), S. F. Reid, O.B.E. (251174); Temporary Squadron Leaders C. Craig (1849), A. T. Roberts (1463), J. H. B. Brown, O.B.E., M.C. (1474), J. L. R. Carter (1239), A. B. K. Watkins (2060), T. G. Millar (2081), K. A. McLean, M.C. (1698), H. C. Finn (2079), J. R. Anderson, M.C. (2743), V. N. B. Willis (2872), L. R. Flynn (263940), E. V. W. Pockley (3752), W. T. D. Maxwell (264363), A. S. Anderson (6714), D. C. Trainor (261653), J. B. G. Gibson (277428), A. M. Johnson (267741), B. N. O. Colahan (3225), H. H. Harrison (7270), A. J. Kerr (2906), G. M. Tallent (257739), J. L. Pollock (3216), T. C. Anthony (297497), E. F. Northcroft (266734), L. D. Walters (277535), W. Deane-Butcher (261286), S. B. Forgan (281237), A. E. Khan (262061), P. C. Tresise (297501), J. A. Conquest (267263), D. Wilson (297509); Flight Lieutenants D. F. O'Brien (253180) (Acting Squadron Leader), D. F. Buckle (263865), C. Roe (277273), G. A. Robbie (256865).

Obituary.

ARTHUR MURRAY CUDMORE.

WE regret to announce the death of Sir Arthur Murray Cudmore, which occurred on February 27, 1951, at Adelaide.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Voss, John Henry Edward, M.B., B.S., 1951 (Univ. Sydney), Sydney Hospital, Macquarie Street, Sydney.

Joseph, Farmey Vincent, M.B., B.S., 1947 (Univ. Sydney), Wagga Wagga.

Medical Appointments.

Dr. W. J. Freeman has been appointed official visitor (chairman) to the Lachlan Park Hospital, New Norfolk, Tasmania.

Dr. P. M. O'Flynn has been appointed to the Department of Public Health, New South Wales.

Dr. R. G. Tonkin has been appointed public vaccinator for the Shire of Lowan, Victoria.

Dr. J. A. Keipert has been appointed public vaccinator for the City of Footscray, Victoria.

Dr. J. B. Hewit has been appointed public vaccinator for the Shire of Cranbourne, Victoria.

Dr. L. L. Hoare has been appointed surgical registrar at the Royal Adelaide Hospital, Adelaide.

Dr. D. B. Wherrett has been appointed government medical officer at Kilcoy, Queensland.

The undermentioned have been appointed medical referees under the provisions of Section 97A, Subsection 7, of the *Workmen's Compensation Act*, 1932-1950, South Australia: Dr. K. S. Hetzel, Dr. M. E. Chinner, Dr. F. R. Hone, Dr. E. B. Jones, Dr. G. A. Lendon and Dr. E. McLaughlin.

Dr. J. A. Roberts has been appointed associate medical officer at the Royal Adelaide Hospital, Adelaide.

Dr. E. L. Roberts has been appointed medical superintendent at the Mental Hospital, Beechworth, Victoria.

Dr. J. H. Downing has been appointed public vaccinator for the Shire of Metcalfe, Victoria.

Dr. G. A. Waterhouse has been appointed public vaccinator for the Shire of Euroa, Victoria.

Dr. A. G. Brown has been appointed public vaccinator for the town of Colac, Victoria.

Dr. P. M. O'Flanagan has been appointed resident medical officer for mental institutions in South Australia.

Diary for the Month.

MARCH 20.—New South Wales Branch, B.M.A.: Council Quarterly.

MARCH 21.—Western Australian Branch, B.M.A.: General Meeting.

MARCH 22.—Victorian Branch, B.M.A.: Executive Meeting.

MARCH 28.—Victorian Branch, B.M.A.: Council Meeting.

MARCH 29.—New South Wales Branch, B.M.A.: Annual Meeting.

MARCH 29.—South Australian Branch, B.M.A.: Branch Meeting.

MARCH 30.—Queensland Branch, British Medical Association: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia; Medical Officer, South Australian Railways.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

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